HEMORRHAGE FROM ESOPHAGEAL VARICES
A CLINICAL STUDY OF 104 PATIENTS

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by

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INTRODUCTION

One of the most difficult and frustrating problems in medicine is the management of bleeding esophageal varices. The onset is usually sudden and unexpected, and the precipitating cause is rarely recognized. The diagnosis is difficult to establish with certainty since bleeding from esophageal varices forms only a small percentage of all upper gastrointestinal hemorrhage. (63) The immediate therapy of bleeding varices is often unsatisfactory, and the nature of the underlying liver disease makes definitive surgery hazardous. Suspectibility to shock and ammonia intoxication, and the predilection to impaired blood coagulation combine to cause an extremely high mortality rate.

This problem of portal hypertension and gastrointestinal bleeding has been recognized for more than four centuries. In 1555 Vesalius portrayed the portal system and described a case of bleeding hemorrhoids, which he believed to be dilated veins belonging to that system. (59A) Morgagni, in 1762, reported a case of hematemesis with "polypoid concretions" in the splenic vein and dilatation of the vasa breves. (41A) In the middle of the 19th century Raciborski (49A) and Raikem (49B) postulated that anastomoses develop between the portal and systemic venous systems and that, in patients

with portal vein thrombosis, these dilated collateral veins in the stomach might bleed. Rokitansky (53A) described a fatal case of hematemesis in 1840 in which esophageal varices, which were thought to have been the site of hemorrhage, were seen at autopsy. By the early part of the twentieth century it had become apparent that there existed the triad of splenomegaly, ascites, and esophageal varices which frequently accompanied portal hypertension.

Esophageal varices are collateral veins which develop between the portal and systemic venous systems in order to relieve hypertension in the portal circulation. The varices represent part of an extensive anastomotic circulation which includes in addition retroperitoneal, hepatophrenic, falciform-umbilical, hemorrhoidal and intrahepatic porto-hepatic anastomoses. The esophageal varices are clinically the most important of these venous collaterals for several reasons. Bleeding from them is frequent and massive, and occurs from an area which is difficult to visualize and to treat. Blood escaping from a ruptured varix is decomposed by intestinal bacteria, liberating ammonia, and perhaps other toxic substances which may contribute to ammonia intoxication and hepatic failure. (39)

Once varices have formed in the submucous venous plexus of the esophagus and stomach, a number of factors probably interact to cause them to rupture and bleed. Regurgitation

of acid and digestive enzymes from the stomach may cause peptic esophagitis and even erosion of the varices. Fluctuations of pressure within the esophageal varices depend on the changes of intrathoracic and intra-abdominal pressure which occur with such normal activities as respiration, coughing, and straining at stool. The role of mechanical irritation of esophageal varices by solid food has not been established. The poor support given to these veins by the tissues of the esophagus may contribute to this problem.

While the history is usually the single most important factor in making the diagnosis of bleeding esophageal varices, it may frequently not be helpful. Although, the classic findings of Laennec's cirrhosis may suggest esophageal varices, they are of limited value in the diagnosis of the individual patient with cirrhosis who frequently bleeds from other sites. (21) Laboratory tests, such as the bromsulphthalein retention (BSP) and the blood ammonium concentration, which are often of value in making the diagnosis, are frequently unsatisfactory. (38,42) Cessation of the bleeding during esophageal tamponade with the Sengstaken-Blakemore tube (SBT) is suggestive evidence that the varices are the source of the hemorrhage. Recently, however, it has been reported that bleeding which has been proven to be due to peptic ulcers of the stomach or small intestine, has apparently been controlled by this method. (12) Barium studies are often of acid and digestive enzymes from the stomach may cause peptic esophagitis and even erosion of the varices. Fluctuations of pressure within the esophageal varices depend on the changes of intrathoracic and intra-abdominal pressure which occur with such normal activities as coughing, sneezing, and straining at stool. The role of mechanical irritation of esophageal varices by solid food has not been established. The poor support given to these veins by the tissues of the esophagus may contribute to this problem.

While the history is usually the single most important factor in making the diagnosis of bleeding esophageal varices, it may frequently not be helpful. Although, the classic findings of Laennec's cirrhosis may suggest esophageal varices, they are of limited value in the diagnosis of the individual patient with cirrhosis who frequently bleeds from other sites. (91) Laboratory tests, such as the bromsulphthalein retention (BSP) and the blood ammonia concentration, while are often of value in making the diagnosis, are frequently unsatisfactory. (78,85) Cessation of the bleeding during esophageal tamponade with the Sengstaken-Blakemore tube (SBT) is suggestive evidence that the varices are the source of the hemorrhage. Recently, however, it has been reported that bleeding which has been proven to be due to peptic ulcer of the stomach or small intestine, has occasionally been controlled by this method. (7) Barium studies are often technically difficult to do and to interpret in a patient with massive bleeding. (63) Some authors are reluctant to use this method in the acute stages of bleeding because the manipulations involved may aggravate or reinitiate the hemorrhage. (16) The surest way of confirming the diagnosis of bleeding varices is with direct visualization by esophagoscopy. (63) However, this requires experienced observers who may sometimes be unable to ascertain the site of bleeding in the presence of massive hemorrhage.

The treatment of patients with bleeding varices is a highly controversial subject. Prior to 1930 the only available treatment consisted of occasional blood transfusions, sedation, and bed rest. About 1930 esophageal tamponade was first used by Westphal with a crude single-lumen tube. (66) The SBT, which was introduced in 1950, is the most efficient and widely employed of the various tubes which have been tried. (9, 51) Although most authors report that the SBT controls the bleeding from esophageal varices it is difficult to be sure how much its use has affected the over-all mortality. Only a few large series of patients in whom the tube was used appear in the literature and these are incompletely documented. (43, 51, 65) The various definitive operations that have been devised both to stop the bleeding and to reduce portal hypertension are all major surgical procedures. Transesophageal ligation of the varices and

partial resection of the stomach and esophagus are used to control the bleeding directly. Porta-systemic anastomoses, splenectomy and arterial ligation were designed to prevent hemorrhage from esophageal varices by reducing the pressure within the portal vein. These patients are particularly poor candidates for major surgery because of the very frequent association of poor liver function. (11, 18)

It has become apparent that patients at the Grace-New Haven Community Hospital with bleeding esophageal varices have had an extremely high mortality. This study was undertaken to review the overall experience at this hospital with particular reference to the efficacy of esophageal tamponade, and to evaluate the survival rate in relation to the form of therapy employed. The main purpose of this review is to outline a diagnostic and therapeutic program which, in the light of our experience, is most likely to lead to a successful outcome.

METHOD

The data in this study were obtained from the Record Room files of the Grace-New Haven Community Hospital, and from the recorded autopsy findings of the Department of Pathology. The fifteen years between January 1, 1943 and December 31, 1957 are included. In this way, 1950, the year partial resection of the stomach and esophagus was used to control the bleeding directly. Portal-systemic shunt procedures, splenectomy and arterial ligation were designed to prevent hemorrhage from esophageal varices by reducing the pressure within the portal veins. These patients were subsequently poor candidates for major surgery because of the very frequent association of poor liver function. (15, 16)

It has become apparent that patients at the Grace-New Haven Community Hospital with bleeding esophageal varices have had an extremely high mortality. This study was undertaken to review the overall experience at this hospital with particular reference to the efficacy of emergency diagnosis, and to evaluate the survival rate in relation to the form of therapy used. The main purpose of this review is to outline a diagnostic and therapeutic program which, in the light of our experience, is most likely to lead to a successful outcome.

METHOD

The data in this study were obtained from the Record Room files of the Grace-New Haven Community Hospital, and from the recorded autopsy findings of the Department of Pathology. The fifteen years between January 1, 1940 and December 31, 1954 were included. In that year, 1954, the name in which the Sengstaken-Blakemore tube (SBT) was introduced, marked the midpoint in the study and this permitted the observation of equal periods of time both before and after its introduction.

The charts examined were those of all adult patients in whom the diagnoses of Laennec's cirrhosis, coarse nodular cirrhosis, primary biliary cirrhosis, obstructive biliary cirrhosis, congestive splenomegaly, and varices of the esophagus were recorded during the fifteen year period. Pediatric cases were not included. Patients admitted while this study was in progress were followed during their hospital course. The patients were treated on both the ward and private medical and surgical services by many private physicians and members of the house staff.

The criteria for inclusion in this series were as follows:

1.) Massive upper gastrointestinal hemorrhage.
2.) Demonstration of esophageal varices by X-ray, esophagoscopy, or post-mortem examination.
3.) Exclusion of other sites of bleeding by history, X-rays, esophagoscopy, gastroscopy, exploratory laparotomy, and post-morten examination.

Patients were excluded from consideration if the gastrointestinal hemorrhage was not severe enough to warrant transfusion of at least 1000 cc. of whole blood, or if other possible sites of bleeding were found or were not adequately eliminated.

RESULTS

One hundred and four patients satisfied the criteria outlined during a total of 130 hospital admissions. Esophageal varices were demonstrated in 93 of these patients. (Table I) In 11, esophageal varices were not confirmed but appeared to be the site of bleeding on clinical grounds. Each of them had cirrhosis of the liver without clinical, radiologic or pathologic evidence of any other site of bleeding.

It is customary, and quite helpful, to divide causes of portal hypertension into two groups -- intra-hepatic block and extra-hepatic block. The former refers to portal obstruction caused by processes within the liver itself, such as cirrhosis, while the latter is due to other causes usually vascular thrombosis either in the portal vein or in one of its branches such as the splenic vein. Histologic confirmation of the diagnosis was obtained in 78 of the 104 patients by liver biopsy, splenectomy or post-mortem examination (Table II). Cirrhosis was diagnosed on clinical grounds in all of the other 26 patients. Thus, it was possible to attribute the portal hypertension to an intra-hepatic block in 95 patients (91%) and to an extra-hepatic block in 9 patients (9%).

The patients ranged between 16 and 82 years of age. However, only 19% were under 40, and only 28% under 50. Seventy-two percent were over the age of 50. The average

TABLE I

Method of Diagnosis of Esophageal Varices

GI Series

GI series only	30 patients
GI series and esophagoscopy	12
GI series and autopsy	12
GI series, esophagoscopy, and autopsy	4
TOTAL	58 patients or 56%

Esophagoscopy

Esophagoscopy only	4 patients
Esophagoscopy and GI series	12
Esophagoscopy and autopsy	5
Esophagoscopy, GI series, and autopsy	4
TOTAL	25 patients or 24%

Transesophageal ligation	1 patient

Autopsy

Autopsy only	26 patients
Autopsy and GI series	12
Autopsy and esophagoscopy	5
Autopsy, GI series and esophagoscopy	4
TOTAL	47 patients or 45%

TABLE II

Diagnosis as established histologically (78 cases)

INTRA-HEPATIC:

Laennec's cirrhosis	40 patients
Post-necrotic cirrhosis	9
Laennec's cirrhosis with hepatoma	5
Biliary cirrhosis	4
Cirrhosis of unknown etiology	4
Sarcoidosis of liver	3
Hemochromatosis	2
Schistosomiasis	1
Laennec's cirrhosis with post-necrotic cirrhosis	1
TOTAL	67 patients

EXTRA-HEPATIC:

Banti's disease	5 patients
Portal vein thrombosis	2
Malignant disease of liver	2
TOTAL	9 patients

age of patients in the extra-hepatic group was 40, with a range between 16 and 65. Forty-five percent of these latter patients were above the age of 50. Fifty-nine percent of the 104 patients were males, and 41% were females. The male and female groups were roughly comparable in regard to diagnosis, clinical features and methods of therapy. All of the patients were white except for 6 Negroes and 1 Puerto Rican.

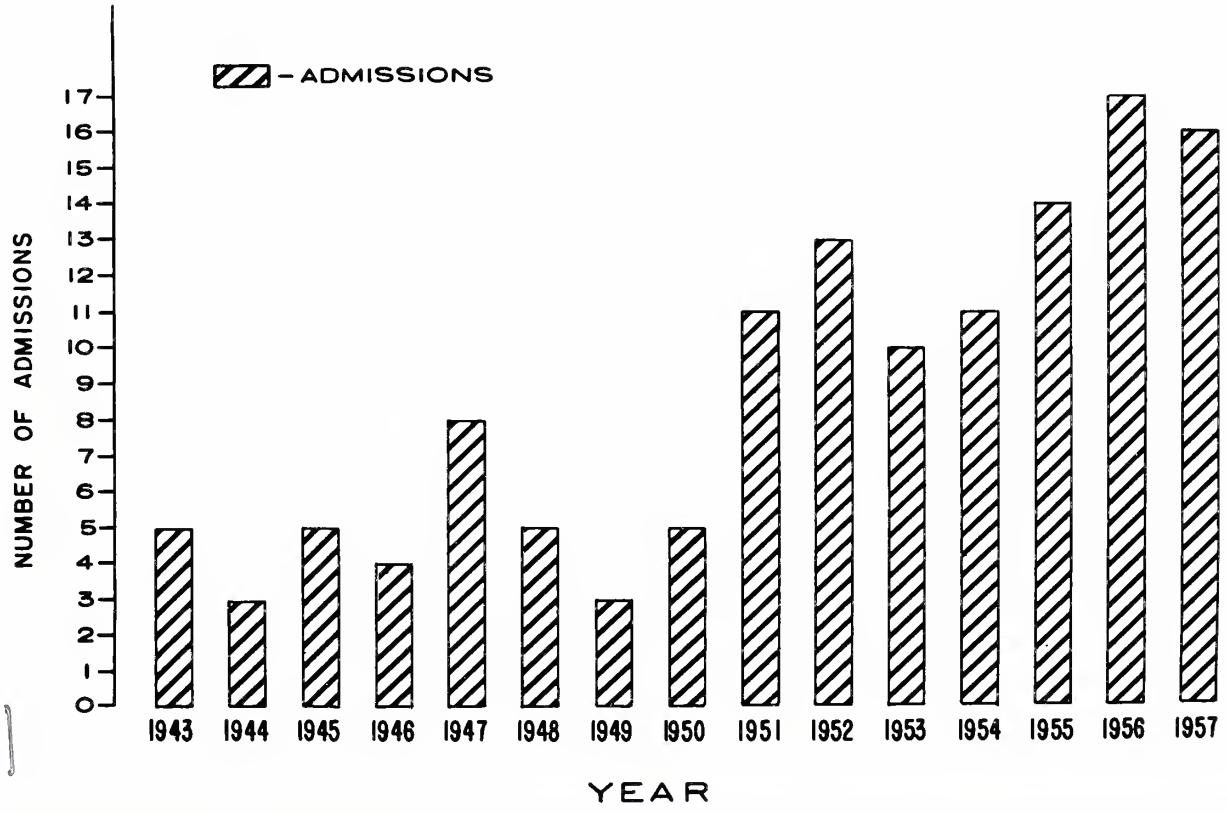
The yearly distribution of the cases is depicted in Fig. I. Thirty-three cases occurred prior to 1950 and the remainder thereafter. This distortion of distribution, despite the fact that equal periods of time both before and after 1950 were reviewed, reflects primarily the incompleteness of our records before 1949.

Severity of bleeding. - An arbitrary system was used to categorize severity of bleeding in these patients. If the patient received up to 2 pints of whole blood during his course of therapy his case was considered mild; if between 2 and 4 pints, then moderate. Patients requiring more than 4 transfusions were considered severe. In fatal cases of hemorrhage the bleeding was estimated to be severe regardless of the amount of whole blood transfused. Sixty-five percent of the 130 cases were severe, 22% were moderate, and 13% were mild.

Clinical condition exclusive of severity of bleeding. - The patient's clinical status was classified as good, fair or poor

FIG. I.

YEARLY DISTRIBUTION OF ADMISSIONS



for each episode of hemorrhage depending on the clinical and laboratory findings. The presence of jaundice, ascites and impending hepatic coma were the principal clinical features considered in evaluating each patient. Liver function tests were independently evaluated with emphasis on serum bilirubin, BSP retention, and serum albumin levels. An overall estimate of the underlying hepatic status of the patient was based on these considerations. This is admittedly not quantitative, but does serve the purpose of roughly classifying these patients. No mere average of the major parameters discussed above was taken to categorize each patient, but rather, these served as guideposts in the overall evaluation of the individual. In 45% of the 130 bleeding episodes the patient was classified as poor, in 29% as fair, and in 26% as good.

Mortality. - The overall mortality for the 104 patients was 72%. This figure is not significantly altered by exclusion of the 11 patients in whom esophageal varices were not actually demonstrated, falling to 70%. Of 38 admissions to the hospital prior to 1951 there were 21 deaths (55%). Of 92 admissions after that year there were 54 deaths (58%). Fifty-seven percent of the 130 admissions ended in death. If a patient ceased bleeding and then started to bleed again while still in the hospital this is counted as one episode. Sixty-eight of the 104 patients died as a result of their first hemorrhage, an incidence of 65%.

In the 95 patients with intra-hepatic block there were 71 deaths (75%). Only 6 of these 71 patients with intra-hepatic portal obstruction survived their first episode of hemorrhage. These patients had an average of only 1.1 episodes of bleeding prior to death. Four of the nine patients (44%) with extra-hepatic block died. These 4 deaths were preceded by an average of 2.3 episodes of bleeding per death. The mortality rates for the various etiologic groups can be seen in Table III. For the whole series the immediate causes of death are shown in Table IV. Yearly mortality statistics are portrayed graphically in Fig. II. It is significant to note that the mortality rate remained relatively stable as the admission rate increased.

Some of the deaths had overlapping causes. However, from the figures in Table IV it is apparent that hemorrhage was responsible for or contributed significantly to 63% of the deaths. Similarly, hepatic coma contributed to 32% of the deaths, and aspiration to 27% of the deaths.

<u>Results of conservative therapy</u>. - Thirty patients, during 46 admissions to the hospital for bleeding received conservative therapy consisting of blood loss replacement, bed rest, sedation and medical management. Esophageal tamponade was not used in these patients and no surgical procedures were performed. Seven patients who died before any treatment including transfusions could be administered are not included

FIG. II

ADMISSION AND MORTALITY STATISTICS 1943 - 1957

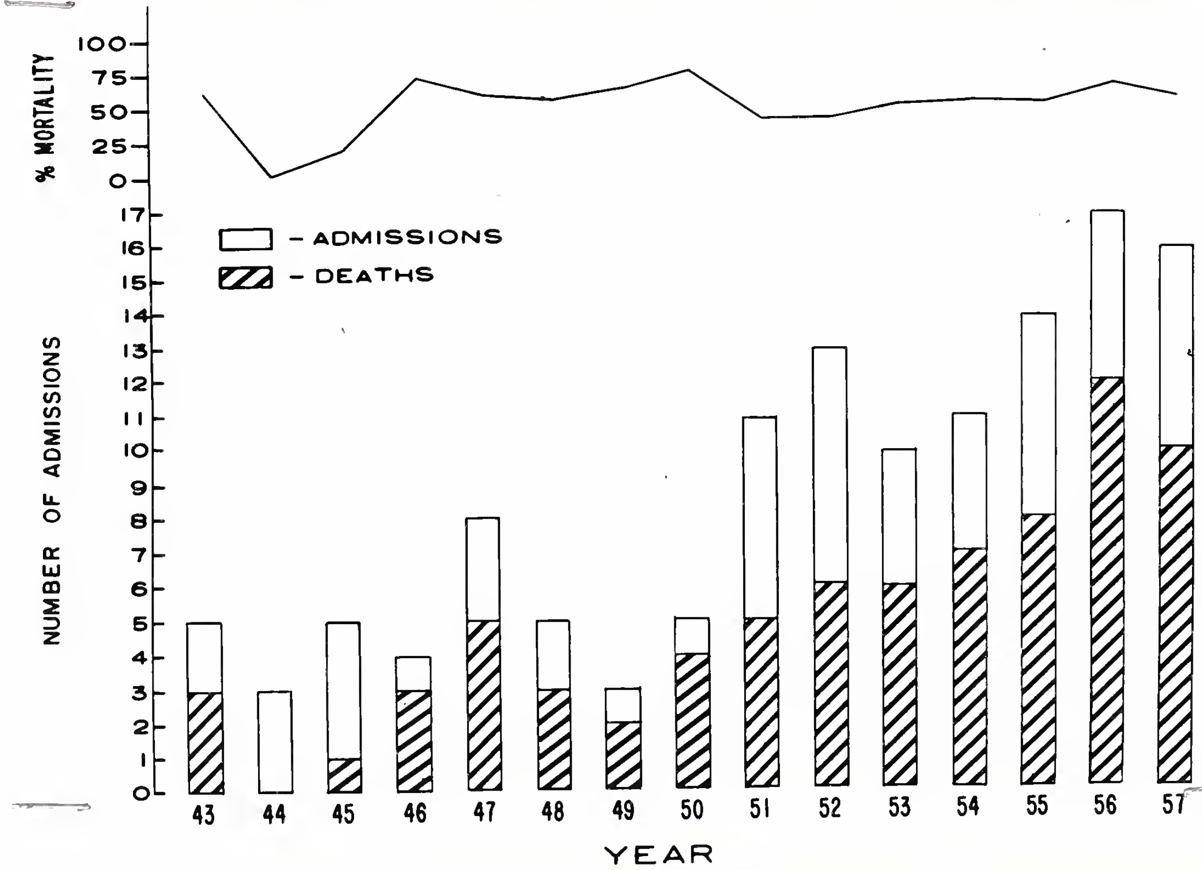


TABLE III

Mortality Rates by Etiologic Group (clinical and histologic)

Diagnosis	Number of Cases	% mortality
INTRA-HEPATIC		
Laennec's cirrhosis with hepatoma	5	100
Hemochromatosis	2	100
Biliary Cirrhosis	7	86
Laennec's cirrhosis	60	77
Cirrhosis of unknown etiology	4	75
Post-necrotic cirrhosis	13	69
Sarcoidosis	3	0
Schistosomiasis	1	0
EXTRA-HEPATIC		
Portal Vein Thrombosis	2	50
Malignant liver disease	2	50
Banti's disease	5	40

TABLE IV

Immediate Cause of Death

HEMORRHAGE (Total 63% of deaths)

Hemorrhage alone	23	patients
Hemorrhage and aspiration	14	
Hemorrhage and hepatic coma	10	

ASPIRATION (Total 27% of deaths)

Aspiration alone	4	patients
Aspiration and hemorrhage	14	
Aspiration and hepatic coma	2	

HEPATIC COMA (Total 32% of deaths)

Hepatic coma alone	11	patients
Hepatic coma and hemorrhage	10	
Hepatic coma and aspiration	2	
Hepatic coma with septicema	1	

MISCELLANEOUS (Total 13% of deaths)

Infection (pneumonia)	2	patients
Operative shock	1	
Pulmonary edema	6	
Respiratory failure	1	

in this group. Although it might be expected that almost all of these patients were admitted prior to 1950, it is interesting to note that 44% of them were treated for bleeding with this type of management after the SBT had become available.

Twenty-one of these 30 patients died (70%). Forty-six percent of the 46 admissions to the hospital ended fatally. Eighty-one percent of the deaths were due completely or in part to hemorrhage. Hepatic coma and pulmonary aspiration each contributed to 24% of the deaths. The 21 patients who died, survived an average of 12.4 days after the onset of bleeding. If the 7 untreated patients, referred to above, are added to these 21 deaths, then the average survival time is reduced to only 10.3 days from the onset of bleeding. The overall clinical condition of this group of patients, as estimated from the criteria described previously, was good in 14%, fair in 43%, and poor in 43% (Figure 4). No deaths occurred in the "good" group. Sixty-two percent of the 21 deaths occurred in the "poor" group, and the remaining 38% in the "fair" category.

The 30 patients who were treated conservatively received an average 2650 cc. of whole blood per bleeding episode. Those who survived averaged 2400 cc., and those who died 2850 cc. There is no significant correlation between the amount of blood administered and survival. The plan of therapy for each of these patients varied widely and

included the use of intravenous saline solutions, Trendelenberg position, vitamins, bland diets, antacids, antibiotics and enemata. These aspects of therapy were not specifically quantitated.

Results of therapy with the SBT. - The SBT was used in fifty patients on 72 occasions. On each of these occasions the tube was left in place for periods from a few moments to 360 hours. Instances in which the tube was deflated and then re-inflated without first being removed were counted as a single usage. The tube was used as many as 5 times during one bleeding episode. However, in over 80% of bleeding episodes it was used only once.

Criteria used to determine whether or not the tube controlled bleeding were based on the disappearance of guiac positive material from the gastric aspirates and stability of hemoglobin and hematocrit levels. Persistence of tarry stools was not felt to be indicative of active bleeding as it is well known that these may continue for many days after the hemorrhage has stopped.

The SBT appeared to control the bleeding on 72% of the 72 individual occasions on which it was used. There were 38 deaths among these 50 patients, a mortality of 76%. The average time from onset of bleeding to death was 15 days. Use of the tube was begun earlier in the episodes of those patients who survived, averaging 41 hours after the onset of

bleeding, as compared to 50 hours in the 38 who died. This does not appear to be a statistically significant difference. The average period of inflation per occasion was 81 hours, and the average total duration of inflation per patient was 110 hours, ranging from a few moments to 15 days. These patients received an average of 4600 cc. of whole blood. Those who survived received an average of 4850 cc., while those who died received 4500 cc.

Of the 38 deaths in this group, hemorrhage, either alone or in combination with other factors appeared to be immediately responsible for death in 45% of the cases. Similarly, hepatic coma was a factor in 42%, and respiratory complications, such as aspiration, in 29%. Indeed, the only 4 deaths in the entire series due to the aspiration of regurgitated gastric contents occurred in patients in whom the tube was being used, and in each of these cases death could be directly attributed to use of the tube.

Complications associated with the SBT occurred in 46% of the 72 occasions on which it was used. Forty percent of these complications were mechanical difficulties such as rupture of either or both of the balloons. Thiry-one percent were respiratory complications including instances of aspiration or airway obstruction related to the tube. Eighteen percent of the complications were pressure effects including erosions, ulcerations, and lacerations of the esophageal

mucosa. These were mainly in the distal esophagus and were discovered at post-mortem. The remaining complications included instances in which the patient regurgitated the tube or else pulled it out voluntarily. The wide variety of miscellaneous difficulties encountered during esophageal tamponade have recently been reviewed and will not be discussed in detail here. (12)

Five patients appeared to have died as direct results of using the SBT. They are presented briefly.

Case 32. - This was a 57 year old white female with Laennec's cirrhosis who had a massive hematemesis while in the hospital for hepatic decompensation. The SBT was inserted immediately and seemed to control bleeding. On the evening of the 4th day the patient appeared cyanotic with severe respiratory distress. The SBT was deflated, followed by regurgitation of large amounts of brownish material and fluid. Aspiratiom was obvious and although the trachea was suctioned the patient died immediately.

Case 52. - This was a 57 year old white male with Laennec's cirrhosis admitted because of massive hematemesis. The SBT was inserted 5 hours after admission, and seemed to control the bleeding. Twenty hours after admission while endotracheal suctioning was being carried out the patient vomited 200 cc. of gastric contents which he aspirated. He died immediately.

Case 53. - This was a 56 year old white female with post-necrotic cirrhosis admitted for hematemesis. The SBT was immediately inserted, and appeared to control bleeding. On the 8th day it deflated spontaneously and she aspirated some vomitus. She became cyanotic and in spite of a tracheotomy she died later that day of aspiration and hemorrhage.

Case 63. - This was a 56 year old Negress with Laennec's cirrhosis. She was admitted for hematemesis. The SBT was inserted shortly after admission. Within 2 days the patient had 4 episodes of respiratory distress. After the last one she became apneic and died. The gastric balloon was found to have burst. Aspiration products were found in the major bronchi at autopsy.

Case 84. - This was a 47 year old white female with Laennec's cirrhosis admitted because of impending hepatic coma and guiac positive stools. The SBT was passed with difficulty and the patient immediately vomited it up and went into respiratory obstruction. In spite of a tracheotomy she died. The immediate causes of death were respiratory obstruction and hemorrhage.

In 3 other patients the SBT probably contributed greatly to death.

Case 14. - This was a 42 year old white woman with generalized hemochromatosis admitted for a cholycystectomy. While in the hospital she went into hepatic coma and then vomited blood. The SBT appeared to stabilize bleeding. On the following day the pressure in the esophageal balloon was found to have dropped spontaneously. The tube was re-adjusted. Three days later it was discovered that she could swallow fluids around the tube and it was removed. She died the next day. Ruptured esophageal varices and fluid blood in the tracheobronchial tree were found at autopsy.

Case 39. - This was a 74 year old Negress with Laennec's cirrhosis admitted for hematemesis. The SBT was immediately inserted but 600 cc. of almost pure blood was vomited around the tube. Thereafter, the aspirate was almost consistently negative for blood. On the 9th day she pulled the tube out herself, but this did not seem to precipitate any further bleeding. The SBT was reinserted,

but the gastric balloon broke and so another tube had to be placed. Again the gastric balloon broke, and another tube was placed. On the 14th day the gastric balloon broke again. During passage of another tube the patient died. Ruptured esophageal varices were seen at autopsy. No mucosal trauma due to the tube was found. This patient had severe respiratory distress on several of these complications due to the SBT.

Case 43. - This was a 53 year old white man with Laennec's cirrhosis admitted from another hospital for vomiting blood. He had been treated with the SBT prior to admission here. He continued to bleed despite the tube, and became comatose. A tracheotomy was performed because of aspiration. On the 2nd day the esophageal balloon presented at his mouth, he became cyanotic and his course began to deteriorate rapidly. Ruptured esophaegeal varices and aspiration pneumonitis were seen at autopsy.

In addition to these serious complications many minor difficulties occurred which did not appear to be critical. These minor difficulties may directly precipitate fatal complications or they may more subtly contribute to death by prolongation or reinitiation of hemorrhage in these very susceptible patients.

Thirteen percent of the patients in whom the SBT was used were felt to be in good clinical condition, 30% in fair condition and 57% in poor condition. None of the deaths were in the good group. Seventy-four percent of the deaths were in the poor group, while 26% were in the fair group. (Figure 4) The bleeding was severe in 81% of cases and moderate or mild in the others. However, this reflects only the amount of blood given during therapy.

Other types of esophageal tamponade. - Nine patients were treated with other types of intra-esophageal tubes, such as the Rowntree (55A) tube. All of these patients, who were treated prior to 1950, died. Only one died of hepatic coma, the others of hemorrhage or a combination of hemorrhage and aspiration. These earlier tubes appeared in general to be unsuccessful in controlling the hemorrhage from esophageal varices.

Transesophageal ligation. - Seven of the 8 patients subjected to this procedure died. The sole survivor was the only patient in this group with an extra-hepatic obstruction. The others were all cirrhotic patients. Although 6 of the 7 who died had post-operative hepatic coma, this was the primary cause of death in only 1 patient. Recurrent hemorrhage was the cause of death in 5 patients, and respiratory failure in the sixth. One of the patients who ultimately died survived for 73 days post-operatively. She died 12 days after a splenorenal shunt was constructed. The other patients lived an average of 7 days after transesophageal ligation. Pre-operative bleeding in all 8 patients was severe.

Prior therapy with the SBT was carried out in 7 patients, including the one who survived. Bleeding was controlled in this manner in 4 patients. One of the patients in whom it did not control bleeding was the patient who survived. Five

patients, including the latter, were bleeding at the time of operation.

Six of the seven patients who died were judged to be in poor clinical condition, and 1 was in fair condition. The survivor was in good condition, although he was actively bleeding at the time of operation. Four patients had ascites at the time of operation, 7 had icterus, and 4 had both. The average serum bilirubin in this group was 4.6 mg. per 100 cc.; serum albumin, 2.5 grams per 100 cc.; BSP retention, 39%.

Post-operative bleeding occurred in 6 patients.

Data on these patients and on shunted patients are presented in Tables V and VI.

<u>Treatment with sclerosing injections</u>. - Six patients received sclerosing injections of their esophageal varices. Almost none of these patients received injection therapy during the acute phase of bleeding.

It is impossible to evaluate this small group of patients, who varied widely in their clinical histories and of whom two-thirds died of other causes.

<u>Portacaval shunts</u>. - Thirteen patients had portacaval shunts and 2 of these died within 14 days. The others left the hospital and at least 8 are still living and well. The surviving pateints were followed for periods of from six

TABLE V

TRANSESOPHAGEAL LIGATION

Case No.	SBT	SBT controlled Bleeding	Clinical Condition	Severity of Bleeding	Post-op. Bleeding	Outcome
4	yes	yes	poor	severe	yes	died
21	yes	yes	poor	severe	yes	died
37	yes	no	poor	severe	yes	died
38	yes	yes	fair	severe	no	died
54	no	-	poor	severe	no	died
60	yes	no	good	severe	no	alive
79	yes	yes	poor	severe	yes	died
81	yes	no	poor	severe	yes	died

TABLE VI

SPLENORENAL SHUNTS

Case No.	SBT	SBT controlled Bleeding	Clinical Condition	Severity of Bleeding	Post-op. Bleeding	Outcome
15	no	-	good	severe	no	alive
50	yes	yes	fair	severe	no	died
60	yes	no	good	severe	no	alive
77	yes	yes	good	severe	no	alive
79	yes	yes	fair	severe	yes	died
80	yes	yes	good	moderate	yes	died
88	yes	no	good	severe	no	alive
90	no	-	good	severe	no	alive
104	no	-	good	moderate	no	alive

TABLE VI (Continued)

PORTACAVAL SHUNTS

Case No.	SBT	SBT Controlled Bleeding	Clinical Condition	Severity of Bleeding	Post-op. Bleeding	Outcome
5	yes	yes	fair	severe	no	alive
22	yes	yes	fair	severe	no	alive
31	no	-	good	severe	no	alive
35	no	-	fair	severe	yes	alive
36	no	-	good	severe	no	alive
41	yes	yes	poor	severe	no	died
67	yes	no	poor	severe	yes	died
69	yes	yes	good	severe	no	alive
83	no	-	good	severe	no	alive
87	no	-	good	moderate	no	alive
92	no	-	good	moderate	no	alive
102	yes	yes	good	moderate	no	alive
103	yes	no	good	severe	no	alive

months post-operative to seven years. However, five of the patients had their operation in 1957 and six had theirs in 1956. Thus, the potential period for follow-up was short in most of the patients. None of these patients had other types of operations designed either to control bleeding or to lower portal hypertension. The shunts were done an average of 17 days after the onset of bleeding, the range being from 5 to 46 days. All of these shunts may be considered as "urgent," since none were emergency and none were strictly elective. "Urgent" shunts are those performed on patients who are not in satisfactory clinical condition for operation, but for whom temporizing would be disastrous. Bleeding was severe in 10 of the cases, including the 2 who didn't survive, and moderate in 3. The cause of the portal hypertension was intra-hepatic in 12 of the patients. The other patient had Banti's disease. The 2 deaths occurred in the former group.

Prior therapy with the SBT was carried out in 7 patients, including the 2 who died. Bleeding was controlled in this manner in 5 patients and was not in the other 2 patients. One of the latter did not survive. Although the tube controlled the bleeding in one of the patients who died later, both of these patients were bleeding at the time of operation.

Six patients, including one who died, had ascites at the time of operation. Four patients, including both of

those who died, had icterus at the time of operation. Three patients, one of whom died post-operatively, had both ascites and icterus at the time of operation. Average total serum bilirubin in 10 of these patients was 2.96 mg. per 100 cc with a range from 0.7 to 7.6. Values were not obtained in 3 patients. Serum albumin in 13 patients was 2.7 grams per 100 cc with a range from 1.9 to 3.2. Average BSP retention for 13 patients was 23% with a range from 4 to 41. The overall clinical condition of these patients was determined in the manner mentioned above. All but the two patients who died were judged either good (8 patients) or fair (3 patients).

Only 2 patients had post-operative bleeding. Post-operative hepatic coma was present only in the two patients who died.

Splenorenal shunts. - Nine patients had splenorenal shunts and 3 of these died. Two of the deaths occurred 12 and 21 days after the operation respectively. The third death occurred 5 years after an elective splenorenal shunt. This patient (Case #80) had Laennec's cirrhosis and died from a second episode of bleeding varices; the immediate cause of death was hemorrhage and hepatic coma. At post-mortem the shunt was found to have been obliterated by a thrombus.

The follow-up period in 5 of the surviving patients

ranged from 2 to 7 years. The other surviving patient was operated on late in 1957. She was the one patient (Case #104) in the entire series who did not enter the hospital because of bleeding. She had bled 3 months previously and was admitted for an elective shunt.

Five of the 9 patients in this group had had previous surgery. In one a portacaval shunt had been attempted but the portal vein was thrombosed. This patient died 21 days after the splenorenal shunt was done. Two patients had previous transesophageal ligation of their varices. One of these died 12 days following the shunt. The other, a patient with Banti's disease, was the only patient in the series who had a transesophageal ligation and survived. One patient had had a splenectomy 21 years prior to his splenorenal shunt which was accomplished with a venous graft. This was the patient with portal, vein thrombosis who had 21 episodes of bleeding. The fifth, a patient with schistosomiasis, had an esophago-gastrectomy at the same operation in which her splenorenal anastomosis was done.

The shunts, excluding the 2 elective procedures, were done an average of 27 days after the onset of bleeding, the range being from 5 to 54 days. Bleeding was severe in the 7 non-elective cases. The cause of the portal hypertension was intra-hepatic in 6 of the patients, and extra-hepatic in the remaining 3. The 3 deaths occurred in the former group.

Of the non-elective patients prior therapy with the SBT was carried out in 5, including 2 who died. Bleeding was controlled in this manner in 3 patients, while in the other 2 it was not. The tube controlled the bleeding in the 2 patients who died later. Only 1 patient was bleeding at the time of operation and this occurred in one of the patients in whom the SBT controlled the bleeding previously and who died.

Two patients, both of whom died, had ascites at the time of operation. Three patients, one of whom died, had icterus at the time of operation. The only patient who had both ascites and jaundice died. Average total serum bilirubin in 7 of these patients was 3.1 mg. per 100 cc. Average serum albumin in 8 patients was 3.3 grams per 100 cc, with a range from 1.7 to 5.0. Average BSP retention for 8 patients was 23%, with a range from 5 to 45. The overall clinical condition of these patients was determined in the manner mentioned above. All patients were judged either good or fair.

Only 2 patients had post-operative bleeding. One of these died 12 days post-operatively, and the other 5 years post-operatively. The other death in this group was caused 21 days post-operatively by pneumonia and shock without a recurrence of bleeding. Post-operative hepatic coma was seen in only one patient who died.

Other surgical therapy. - Nine patients underwent forms of surgery other than shunting procedures and direct ligation of the varices. There were 4 splenectomies, 2 esophago-gastrectomies, 2 splenic and hepatic arterial ligations, and 1 combined hepatic artery ligation with splenectomy. Their histories will be presented briefly.

Case 11. - This was a 32 year old white woman with Banti's disease whose bleeding was controlled by the SBT. Splenectomy was performed on the 11th day with the SBT in place. She was discharged, but returned 16 months later and in spite of an esophago-gastrectomy died of hemorrhage and hepatic coma 2 days post-operatively.

Case 19. - This was a 62 year old white man with Laennec's cirrhosis and a previous episode of variceal bleeding which he survived with conservative therapy. The second episode was 5 months later. He was treated in the same fashion, and 2 months later his hemoglobin had stabilized, and he underwent a hepatic and splenic arterial ligation. He died in pulmonary edema and coma post-operatively.

Case 24. - This was a 55 year old white woman with Laennec's cirrhosis and a 10 month history of decompensated liver disease. One month before the onset of bleeding she underwent a splenectomy. One month later she began to bleed, but the SBT controlled the hemorrhage and the patient was discharged 43 days after the onset.

Case 26. - This was a 53 year old white man with Banti's disease. Splenectomy was performed. In the next 2 years he had 4 bouts of hematemesis, and finally died of hemorrhage. He had had no bleeding prior to splenectomy.

Case 70. - This was a 45 year old white woman with sarcoidosis. Five years prior to admission she had a hematemesis at another hospital and an emergency splenectomy with hepatic artery ligation was carried out. On admission to this hospital 5 years later she had large tarry stools. She was treated conservatively and discharged.

Case 77. - This was a young white man with Banti's disease. At age 9 he had a splenectomy. Subsequently he had 21 episodes of bleeding from esophageal varices. He is still alive.

Case 82. - This was a 52 year old white man with cirrhosis of unknown etiology. Hemorrhage was well controlled with the SBT, and 16 days after the onset a hepatic and splenic arterial ligation was done. Patient was discharged 2 months later.

Case 88. - This is a 33 year old Puerto Rican woman with schistosomiasis. Seven months prior to admission she had a gastrotomy at another hospital in an attempt to discover the source of her bleeding. She was placed on an ulcer regimen. On admission to this hospital she was vomiting bright red blood. The SBT failed to control the bleeding. Five days after the onset of this second episode of bleeding she underwent a splenorenal anastomosis and simultaneous esophago-gastrectomy. She is alive 5 years later.

It is obvious that this is a small group of patients with heterogeneous aspects, both with regard to etiology and to therapy. It is doubtful that any conclusions can be made concerning these surgical procedures and their effectiveness.

Hepatic coma. - Aside from hemorrhage, hepatic coma was the single most important cause of death in this series of patients.

It is necessary to consider it separately because, although its occurrence is ominous, it is only indirectly involved in the problem of acute hemorrhage from varices. There were 48 instances, in the same number of patients, of hepatic coma. In 39 of these cases hepatic coma followed the onset of bleeding, and in the remaining 9 cases the bleeding from varices began during previously existing coma. Four of the 48 cases were in the fair category; the rest were in the poor group. Only 3 patients survived. All 3 had Laennec's cirrhosis and were in poor condition. There were only 2 patients with hepatic coma who did not fall into the intrahepatic group. One of these had Banti's disease. The other had a primary carcinoma of the liver without cirrhosis.

Respiratory disease. - Respiratory problems were common in all of these patients, particularly in the SBT-treated group. Aspiration pneumonitis and atelectasis occurred commonly. The frequency of tracheotomy in these patients emphasizes the respiratory difficulties. Autopsy confirmed the frequency of these problems.

DISCUSSION

The high mortality and the suddenness with which patients may die of hemorrhage from esophageal varices make it an emergency situation of the most serious nature. One

of the major causes of the high mortality (63) in bleeding esophageal varices is the difficulty in establishing an early diagnosis. The medical history is of limited value in determining the site of hemorrhage in upper gastrointestinal bleeding. (63) Either hematemesis or melena may occur. The amount and color of the regurgitated blood is of little diagnostic value. The welling up of blood into the mouth in the absence of nausea is quite unreliable. Similarly the onset of bleeding following coughing or straining at stool is almost worthless as a diagnostic clue suggesting variceal bleeding. The absence of any gastrointestinal symptoms which might suggest peptic disease is of some value. It is well recognized however that gastrointestinal hemorrhage is frequently the first symptom of a peptic ulcer. (1)

A history of previously diagnosed liver disease is strong evidence that the bleeding may come from esophageal varices. The positive history of prolonged heavy alcohol ingestion, chronic malnourishment, previous hepatitis or recurrent disease of the biliary tree suggests that liver disease may be present. However, even in the full blown picture of cirrhosis gastrointestinal bleeding from sites other than esophageal varices is not uncommon, (38) particularly peptic ulcers. Fainer and Halsted, in an analysis of upper alimentary tract bleeding in 76 patients with cirrhosis, found that 18.4% of the patients bled from peptic ulcer. (21)

Or particular importance are the signs of hepatic failure, notably disturbances of consciousness, the characteristic flapping tremor or foetor hepaticus, which are often precipitated by gastrointestinal bleeding in patients with cirrhosis.

There are several laboratory tests which are of some value in the diagnosis. The Bromsulphthalein (BSP) Retention test, which is a measure of both hepatic blood flow and hepatocellular function, is usually abnormally increased in cirrhotic patients. Retention also frequently occurs after massive hemorrhage due to decreased hepatic blood flow and hypoxia. (38, 42) It should be emphasized that this retention can occur after massive hemorrhage from any source and that the test is not specific for bleeding varices. Conversely, patients with extra-hepatic portal obstruction may occasionally have a normal BSP even though bleeding varices are present. (42) Recently, the blood ammonium has been reported to reflect quite accurately the presence of portal-systemic venous collaterals in patients with gastrointestinal bleeding. (38) The blood ammonium is presumably elevated in cirrhotic patients with gastrointestinal bleeding because the collateral circulation enables ammonia, which is liberated in the intestine by the action of bacteria on blood, to bypass the liver and thus achieve high levels in peripheral blood. This determination appears to be more accurate than the BSP in determining

the presence of cirrhosis. Although McDermott (38) implies otherwise, this test does not appear to be able to differentiate bleeding varices from a peptic ulcer in a cirrhotic patient. Blood ammonium determinations were performed in only a few of the patients in this series.

Esophageal tamponade with the SBT is frequently considered to be a reliable means of differentiating between esophageal varices and other sites of gastrointestinal hemorrhage. In this series with known esophageal varices it appeared to control the bleeding in only 72% of the occasions. If one were to assume that continued bleeding in spite of esophageal tamponade represented bleeding from other sites we would have failed to diagnose correctly a large percentage of our patients. Conversely, bleeding from peptic ulcers tends to stop regardless of therapy (1) and this may explain the apparent control by esophageal tamponade of some cases of hemorrhage from peptic ulcers. (12) Finally, a major objection to the immediate use of the SBT as a diagnostic tool is that later esophagoscopy is rendered more difficult by virtue of the pressure effects of the balloon on the anatomy of the mucosa.

Barium studies are especially useful in the diagnosis of lesions of the upper gastrointestinal tract. Some believe that swallowing the barium will increase the hazard of further

bleeding and may damage the mucosa. (16) Others have found that it does not cause any complications and has considerably increased the number of positive findings. (63) Fifty-eight patients (56%) in this series had varices demonstrated on X-ray. However, some of these X-rays were taken before the patient bled (41%), and many of the remaining studies were done after bleeding had stopped (37%). Twenty-two percent (13 patients) of the GI series were done during the early phase of bleeding. There were no instances among these 13 patients of intensified or prolonged hemorrhage.

Weber has stated that "in lesions of the stomach and duodenum, radiography is more revealing than gastroscopy, but in esophageal lesions esophagoscopy is more accurate than radiography." (63) This is well borne out by our own data. Twenty-seven patients had esophagoscopy. Varices were demonstrated in 25 of these, and in the remaining 2 patients, varices were demonstrated at autopsy. Of 12 patients who had both esophagoscopy and GI series there were 3 in whom varices were not seen on the X-rays, but in whom the endoscopic procedure was positive for varices. There were no patients in whom esophageal varices were found by X-ray, but not by esophagoscopy. There was only 1 complication of esophagoscopy in this series. In this patient (Case #49) the esophagus was perforated and she developed an empyema. The patient subsequently died in hepatic coma.

In the presence of severe active bleeding esophagoscopy

may be of little value in determining the origin of the hemorrhage. Even experienced esophagoscopists cannot identify the bleeding site when their field of vision is obscured by blood. On the other hand examination after bleeding has ceased may demonstrate esophageal varices, but it cannot furnish proof that the varices were the cause of the hemorrhage. Although the finding of bleeding from such a varix is proof positive, the presence of an esophageal erosion over or a thrombus within a varix is only circumstantial evidence. In our series of 25 positive esophagoscopies, actual bleeding from a varix was demonstrated in only 11 cases (44%).

The sole universally accepted diagnostic requirement of bleeding esophageal varices is direct visualization of the bleeding varix. Using this as the sole criterion only about 10% of our cases fulfill these requirements. By definition, in order to satisfy this rigid criterion, esophagoscopy must be performed while the patient is actively bleeding. All of these 11 patients, none of whom had evidence of aggravation of bleeding were examined within the past few years.

The dangers of esophagoscopy range from causing recurrent bleeding to perforation. The precipitation of regurgitation and bronchial aspiration can be avoided by the placement of an inflated tracheal cuff prior to esophagoscopy. The

hazards of esophagoscopy and their prevention are reviewed elsewhere (22) and will not be discussed here.

The treatment of bleeding from esophageal varices usually is classified as medical or surgical. Medical therapy is considered synonymous with conservative therapy. Until recently it consisted principally of blood replacement, sedation and bed rest, and carried with it an awesome mortality (Table VII). Of Higgins' 115 cases treated with transfusions and sedation prior to 1947, 84% died. (26) The introduction of the SBT to the armamentarium seemed to promise a new and successful era in this medical management of variceal hemorrhage. In spite of the apparent success of esophageal tamponade in controlling bleeding, the mortality appears to have been only slightly reduced. (Table VIII) In the series by Welch et al. 2/3 of 19 patients on whom the SBT was used died. (65)

Surgical therapy can be classified into two main categories. In the first the surgical procedures are designed primarily to control acute hemorrhage. Included in this group are transesophageal ligation, and resections of the stomach and/or distal esophagus. The other group includes those procedures devised to reduce the level of pressure within the portal system. Chronologically, they include splenectomy, packing of the mediastinum, hepatic artery ligation and a variety of porta-systemic shunting operations currently in use.

TABLE VII

RESULTS OF CONSERVATIVE THERAPY

AUTHOR	NUMBER OF PATIENTS	MORTALITY %
Higgins (1947)	115	84%
Patek et al. (1948)	42	50% *
Douglass & Snell (1950)	71	50% **
Reynolds et al. (1952)	33	72%
Ratnoff & Patek (1942)	106	70% **
Gould (1958)	30	70%

* - within a year after the first hemorrhage. Some of these had peptic ulcers.

** - within a year after the first hemorrhage. The number of patients with proven varices is not given.

*** - within a year after the first hemorrhage.

TABLE VIII

RESULTS OF TAMPONADE WITH THE SENGSTAKEN-BLAKEMORE TUBE

AUTHOR	NUMBER OF PATIENTS	% OF PATIENTS CONTROLLED	MORTALITY %
Sengstaken & Blakemore (1950)	30	?	?
Reynolds et al. (1952)	32	67%	47%
Nachlas et al. (1955)	14	71%	100% *
Welch et al. (1956)	19	? **	67% plus
Gould (1958)	50	72%	76% plus

* -- The 71% listed above are described in the article as "fair to satisfactory" results.

** -- The tube was said to have stopped the bleeding in the majority of the 19 patients.

Among the emergency operations transesophageal ligation is the most widely employed. Crile (14, 15) and Linton (33, 34) consider it to be therapy of choice. The latter believes that as an emergency homeostatic procedure it may gain time to allow definitive corrective surgery at a more opportune time. Linton has stated that the frequency of recurrent hemorrhage from esophageal varices after transesophageal ligation is too high to consider this operation to be complete therapy. (33) Crile, on the other hand, favors this operation alone, particularly in patients with extra-hepatic portal obstruction. (15) In addition he has little confidence in the ultimate effect of shunting operations. Results of this procedure are recorded in Table IX.

Welch has recently suggested a transabdominal transesophageal ligation. (64) This procedure, which was devised to allow exploration of the duodenal and gastric areas prior to ligation of esophageal varices, has not yet been widely used.

Esophagogastrectomy is a procedure by which the bleeding site can be excised. Some authors advocate its use as the treatment of choice to control bleeding since it removes gastric as well as esophageal varices. (35) This operation has not been performed extensively. (3, 49, 59). The two main disadvantages to the procedure are post-operative digestive disturbances, and the probability of new varices forming at the esophagojejunal junction.

TABLE IX

RESULTS OF TRANSEOPHAGEAL LIGATION

AUTHOR	EXTRA-HEPATIC BLOCK	INTRA-HEPATIC BLOCK	REBLED %	MORTALITY %
Crile (1950 and 1953)	7 patients	-----------	29%	0
Linton (1953)	3 patients	12 patients	33%	13%
Linton & Warren (1953)	2 patients	9 patients	36%	18%
Welch (1956)	----------	5 patients	?	60%
Gould (1958)	1 patient	7 patients	75%	88%

Splenectomy, which was designed to remove the significant contribution of the splenic blood flow to the portal circulation, has been done primarily on patients with Banti's syndrome. (2, 10, 27) It has never been demonstrated conclusively that splenectomy effectively reduced portal pressure permanently in cirrhotic patients, although it may be of some value in patients in whom portal hypertension is caused by Banti's disease or splenic vein thrombosis. In these patients there has been, in general, a high incidence of rebleeding. (19) This operation performs the additional function of correcting splenic pancytopenia when it is present. A review of this material is presented in Table X.

Splenic artery ligation has been done in an attempt to reduce portal hypertension in a manner analagous to splenectomy. It has not been a popular operation and the results have not been promising. (4, 6, 20) Ekman summarized the experience of a total of 14 cases from the literature. (19) He found that rebleeding was common, but that operative mortality was lower than that of splenectomy. Madden ligated both the hepatic and the splenic arteries in 8 cirrhotic patients. (36) Seven of these died. These poor results are consistent with the experiments of Julian and Dye which demonstrated that ligation of the splenic artery is not accompanied by an appreciable drop in portal pressure. (30) Hepatic artery ligation, which has been performed

TABLE X

RESULTS OF SPLENECTOMY

AUTHOR	DIAGNOSIS	NUMBER OF PATIENTS	MORTALITY %	REBLEEDING %
Howells (1938)	Banti's	57 *	? *	100% *
Barg & Dulin (1940)	Banti's	22	27% **	? **
Carlgren et al. (1949)	Banti's	7	? ***	100%

* How many of the 57 bled pre-operatively isn't known. However, all who did, continued to do so after the operation. Mortality isn't given.

** The article states that of 5 patients who had bled pre-operatively, 2 rebled afterwards, and 4 who had not bled previously, did so later on.

*** Mortality not given.

in only a small number of patients has produced indifferent results. (36, 40) Ligation of the coronary vein which is the major link between the portal system and the periesophageal plexus has been tried, but without much success in controlling hemorrhage. (35)

Garlock and Som (23) packed the posterior mediastinum with irritant materials in order to stimulate further collateral channels between the azygos system and the overloaded submucosal plexus of the esophagus. After operation the esophageal varices have appeared to get smaller by radiographic studies. It has been reported in only eight patients and has not been enthusiastically received by other surgeons.

Injection of the esophageal varices with sclerosing solutions was introduced in 1939. (13) It cannot be considered as a good emergency measure or a definitive mode of therapy. A review of this material is presented in Table XI. As a practical means of therapy, however, principally in patients who are considered poor surgical risks, it seems to offer a relatively safe method of treatment. More patients will have to be treated and larger follow-up observations made before injection of varices can be properly evaluated as a form of therapy.

At the present time porta-systemic shunts are in wide use. In one type, the splenorenal, the spleen is removed and the splenic vein is anastomosed to the left renal vein, thus permitting some of the portal blood to enter the systemic

TABLE XI

RESULTS OF INJECTION OF ESOPHAGEAL VARICES WITH SCLEROSING SOLUTIONS

AUTHOR	DIAGNOSIS	NUMBER OF PATIENTS	MORTALITY %
Crafoord & Frenckner (1939)	Banti's	1	0
Patterson & Rouse (1946)	Banti's	2	0
	Cirrhosis	4	0
	Unknown	2	50%
Moersch (1947)	Banti's	19	35%
	"Hepatitis"	3	34%
Macbeth (1955)	Banti's	16	13%
	Cirrhosis	14	50%

circulation. In portacaval shunts the portal vein is divided near the hilum of the liver and the distal segment anastomosed directly to the inferior vena cava, thus allowing some of the portal blood to bypass the liver. In general, splenorenal shunts have been performed in cases of extra-hepatic block because the portal vein has often undergone cavernous transformation, thus making portacaval shunts impossible. In cirrhotic patients, in whom the portal vein is usually patent, the portacaval shunt has been more commonly used. This type of shunt seems to be more effective because it shunts more of the portal blood than the splenorenal. Linton feels that this increased shunting of portal blood in portacaval shunts may result in increased post-operative mortality rates from liver failure. (33) As might be expected however the incidence of post-shunt bleeding appears to be lower after portacaval shunts, the group with the greatest shunting. (19, 29)

Most authors have reported a relatively high salvage rate in shunted patients (Tables XII and XIII) This may in part be due to the fact that the patients usually selected for shunts are in better medical condition than the average patient with bleeding esophageal varices. (11) This fact is probably also responsible for the disproportionate number of patients with extra-hepatic portal obstruction who have had porta-systemic shunts constructed. That is, the percentage of patients with Banti's disease who are shunted is greater

TABLE XII

OPERATIVE MORTALITY AFTER PORTACAVAL SHUNT

AUTHOR	NUMBER OF CASES	NUMBER OF DEATHS	MORTALITY %
	INTRA-HEPATIC GROUP		
Blakemore (1951)	79	16	20*
Large et al. (1952)	12	2	16
Jahnke et al. (1954)	25	2	8
Child (1955)	56	8	14
Ellis et al. (1956)	37 **	15	40
Linton & Ellis (1956)	33	5	15
Walker (1957)	56 **	3	5
Hallenbeck et al. (1957)	29	7	21
Ekman (1957)	29	5	17
Gould (1958)	12	2	16
	EXTRA-HEPATIC GROUP		
Blakemore (1951)	32	?	20*
Large et al. (1952)	3	0	0
Linton & Ellis (1956)	3	0	0
Gould (1958)	1	0	0

* Separate mortality rates for intra- and extra-hepatic groups are not given.

** Most, but not all, of these patients were in the intra-hepatic group.

TABLE XIII

OPERATIVE MORTALITY AFTER SPLENORENAL SHUNT

AUTHOR	NUMBER OF CASES	NUMBER OF DEATHS	MORTALITY %
INTRA-HEPATIC GROUP			
Jahnke et al. (1954)	4	0	0
Ellis et al. (1956)	88 *	21	24
Linton & Ellis (1956)	70	9	13
Ekman (1957)	13	1	8
Gould (1958)	6	2	33
EXTRA-HEPATIC GROUP			
Jahnke et al. (1954)	2	0	0
Ellis et al. (1956)	88 *	21	24
Linton & Ellis (1956)	13	0	0
Paltia & Sulamaa (1955)	8	2	25
Ekman (1957)	21	0	0
Gould (1958)	3	0	0

* The relative number of cases of intra- as opposed to extra-hepatic block is not given.

than the percentage of cirrhotics who are shunted. This also lowers the mortality rate in the total group of shunted patients because patients with extra-hepatic obstruction have a better prognosis, even without definitive treatment. (33)

Most shunts have been constructed as elective procedures after bleeding episodes have subsided. Some have been performed as emergency operations after periods of non-surgical management have failed to controll bleeding. Many patients have rebled during these periods of preoperative observation and for this reason some authors have advocated emergency or "urgent" shunts on all patients in whom bleeding does not quickly stop except for those in whom "hepatic function is such that an early demise is properly predicted even in the absence of hemorrhage." (44)

In Tables XII and XIII are summarized some of the results of shunting operations reported in the literature. It is important to note that in many of these series the preoperative condition of the patients is not specified and therefore the high survival rates must be evaluated with extreme caution. The incidence of rebleeding after shunt operations is shown in Table XIV. In general, rebleeding seems to be more common after splenorenal shunts. This has been attributed to the smaller volume of portal blood

TABLE XIV

FREQUENCY OF HEMORRHAGE AFTER SHUNT OPERATIONS

AUTHOR	SPLENORENAL		PORTCAVAL		LENGTH OF FOLLOW-UP
	Cases	Rebled?	Cases	Rebled?	
Julian & Dye (1951)	17	0	3	1	2 - 30 mos.
Blakemore (1952)	52	6	46	3	1 mo. - 6 yrs.
Jahnke et al. (1954)	6	1	23	1	2 mos. - 3 yrs.
Walker (1954)	-	-	23	3	1 mo. - 3 yrs.
Child (1955)	-	-	48	0	1 mo. - 4 yrs.
Linton & Ellis (1956)	66	13	26	3	1 - 9 yrs.
Ekman (1957)	32	14	20	0	1 - 7 yrs.
Gould (1958)	9	2	13	2	1 mo. - 7 yrs.

that is being shunted, and to the more modest decrement in portal pressure achieved. (19)

Our results in this treatment of these 104 patients have been, in general, similar to other series reported. The overall mortality was 72%. In the group of 30 patients who were treated conservatively without esophageal tamponade 21 died, a mortality of 70%. Esophageal tamponade did not decrease this mortality. In fact the mortality was 76% in the patients in whom the SBT was used. A comparison of these two large groups of patients can be seen in Tables XV and XVI. In the group treated without tamponade there was no difference in mortality between patients seen either before or after 1950, the year which marks the introduction of the SBT.

One would expect a lower mortality when a direct form of therapy, such as esophageal tamponade, is used in addition to all of the other aspects of medical management. However this was not the case. The reasons for the similar mortality in both the tamponaded and non-tamponaded groups are not clear. However, there were more patients in the poor category among those in whom the SBT was used. This alone confers ominous prognostic implications. (See Figure IV) Secondly, 8 of the 38 deaths (21%) in the tamponaded group were due either completely or in part to the tube itself.

TABLE XV

CONSERVATIVE TREATMENT

(30 Patients)

	No. of Patients	%
Severity of bleeding		
severe	15	50%
moderate	12	40%
mild	3	10%
Clinical condition		
poor	13	43%
fair	13	43%
good	4	14%
Diagnosis		
Intra-hepatic group		
Laennec's cirrhosis	20	67%
Laennec's cirrhosis with hepatoma	3	10%
Post-necrotic cirrhosis	1	3%
Biliary cirrhosis	1	3%
Cirrhosis of unknown etiology	1	3%
Sarcoidosis	1	3%
Extra-hepatic group		
Banti's disease	2	7%
Portal vein thrombosis	1	4%

Ultimate definitive therapy

One patient had a splenorenal shunt. There were no other operations in this group after institution of conservative therapy.

TABLE XVI

TREATMENT BY ESOPHAGEAL TAMPONADE (50 PATIENTS)

	No. of Patients	%
Severity of bleeding		
severe	40	81%
moderate	7	13%
mild	3	6%
Clinical condition		
poor	28	57%
fair	15	30%
good	7	13%
Diagnosis		
Intra-hepatic group		
Laennec's cirrhosis	27	54%
Post-necrotic cirrhosis	8	16%
Biliary cirrhosis	4	8%
Hemochromatosis	2	4%
Cirrhosis of unknown etiology	2	4%
Laennec's cirrhosis with hepatoma	1	2%
Metastatic carcinoma of liver	1	2%
Sarcoidosis	1	2%
Schistosomiasis	1	2%
Extra-hepatic group		
Banti's disease	2	4%
Portal vein thrombosis	1	2%

Ultimate definitive therapy

1. Transesophageal ligation was done on 7 of these patients an average of 6 days after the onset. Two of these patients later had splenorenal shunts. One of these died. Six of these seven patients died.

2. Portacaval shunt was carried out on 7 patients an average of 15 days after onset. Two died.

3. Splenorenal shunt was carried out on 5 patients an average of 25 days after onset, and on one patient 5 years after her first hemorrhage. Three of these 6 patients died.

4. A splenectomy, an esophagogastrectomy, an esophagogastrectomy along with a splenorenal shunt, and a splenic and hepatic arterial ligation were performed on other patients in this group.

It is evident however that the tube does control bleeding in the majority of instances. There is even a suggestion that early institution of tamponade may be associated with a higher survival rate. However, the average period of time lapse between the onset of bleeding and the institution of tamponade was only an average of 9 hours shorter in those who survived (50 hours vs. 41). There were 30 instances in which the SBT was passed within the first 24 hours of bleeding. The fact that 21 of these patients (70%) died indicates that early institution of tamponade, by itself, is not attended with a lower morality.

Hemorrhage was the cause of death in 81% of patients treated conservatively, but in only 45% of those treated with the SBT. Hepatic coma as a cause of death was more important in the latter group. This suggests that the tube does control bleeding, but, ironically, its use seems to prolong life to have the patients succumb from hepatic coma rather than from hemorrhage per se. This is certainly an oversimplified statement, but it appears to be a valid clinical impression.

Patients in the tamponaded group received almost twice as much whole blood as the conservatively treated patients. The amount of blood did not affect the mortality rates of the two groups. Since the severity of bleeding was determined by the amount of blood given as transfusions,

it is not a valid point of comparison between the two groups.

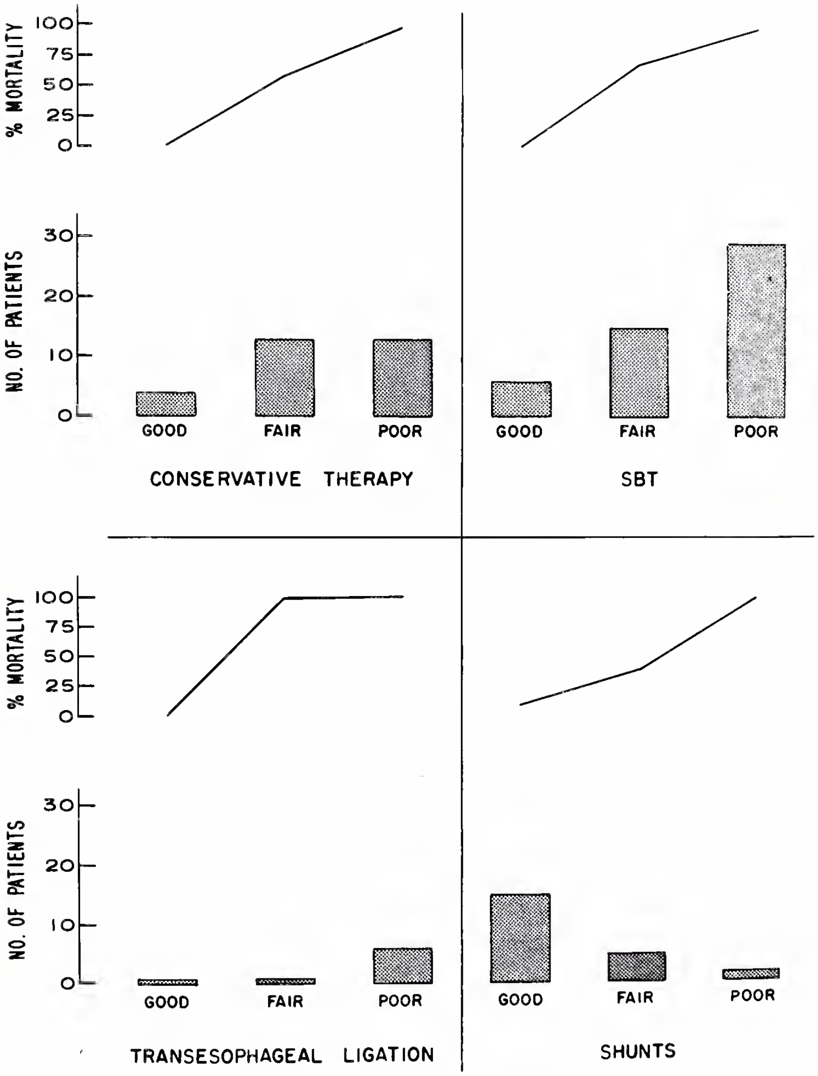
If the high (38%) incidence of complications of the SBT could be decreased it is possible that use of the tube under those conditions might significantly lower the mortality. Most of the serious complications incurred in the use of esophageal tamponade appeared related to incorrect use of the tube or inadequate care of the intubated patient. It appears likely that better use of the SBT can reduce the incidence of complications. (12)

The use of sclerosing solutions was not a frequent method of treatment in our series. It is impossible to evaluate its role in the survival rate of those patients in whom it was used. In general, it has not been used as a definitive method of treatment, but may be of value in selected patients.

In contrast to the high mortality rates in the above groups, the patients who were shunted did very well. There was an overall mortality of 15% among the patients who underwent portacaval shunts, and a mortality of 29% in the 7 non-elective splenorenal shunts. These rates are comparable to those of similar studies in the literature. It is extremely significant that only 2 patients out of 22 who had a shunting procedure were in the "poor" category. (Figure IV) Thus, they were generally in better clinical condition than the patients who were treated medically, and this is undoubtedly

FIG. IV.

MODE OF THERAPY, CLINICAL CONDITION AND SURVIVAL STATISTICS



one of the reasons why they survived not only bleeding from esophageal varices, but also a major surgical procedure. This is made even more clear by the disastrous results we have observed with transesophageal ligation. Seven of the 8 patients who underwent this operation died. Six of them were in "poor" condition pre-operatively, 1 was in "fair" condition, and the lone survivor was in "good" condition.

An attempt was made to determine what criteria could be used to predict the prognosis of these patients. Although the series was relatively evenly divided between males and females, it was found that the latter had a higher overall mortality, 84% as compared with 64% for the males in the group. The reasons for this discrepancy are not evident. The breakdown into specific diagnoses showed an even distribution between the two sexes. However, when the mortality rate is related to the clinical condition of each patient, the sex difference becomes more understandable. Thus, 18% of the males were in the "good" category, and 13% of the females were in that group. Thirty-five percent of the males and 23% of the females were in the "fair" group. Twenty-eight percent of the males and 64% of the females were in the "poor" group. In most other series in the literature the females have had a better survival rate than the males. The reasons for the poorer clinical condition of our female patients is not apparent.

Laboratory criteria for operation in these patients have been suggested in the literature. The absence of ascites, minimal icterus, and a serum albumin of at least 3.0 grams per 100 cc. have been held by both Linton and Child to be desirable before considering major operative procedures such as shunts or transesophageal ligation. (11, 33)

The average serum albumin in the patients with intrahepatic block who survived was 2.7 grams per 100 cc. In those who died it was 2.2 grams per 100 cc. Of those in the surviving group, 35% had levels over 3.0 grams, but in the latter group only 11% had such levels. Thus, the serum albumin level, while it does correlate roughly with survival rates, is not a good indication of prognosis in individual cases. Although a serum albumin of 3.0 grams per 100 cc. or more is certainly desirable, less than 50% of the patients who had shunts constructed fulfilled these criteria. The level was usually considerably below this in patients who did not survive the three operations with which we have had most experience.

The cephalin flocculation test was done on most of the patients. Only four percent of those who died had zero or one-plus values, while 19% of those who did survive had similar levels. This test is a poor prognostic criterion.

The BSP retention test proved to be fairly valuable. A BSP retention of 20% or less was seen in 48% of the

survivors, but in only 23% of those who died. However, the average level in the former group was 26%, and 38% in the latter. Thus, rather high levels were common in both groups, and in individual cases it did not prove to be of prognostic significance. The average level in the group which was shunted was 23%, but in the group undergoing transesophageal ligation it was 39%

The average serum bilirubin in patients who died was 5.8 mg. per 100 cc, and in those who survived was 3.5 mg. per 100 cc. However, the percentage of patients in both groups whose level was 1.5 or less was 25% and 29% respectively. Thus, it wasn't a good prognostic criterion.

In our hands the best prognostic criterion in these 104 patients was an evaluation based on a composite picture of the presence or absence of ascites and jaundice, the state of several liver function tests (BSP and serum albumin), and the presence or absence of hepatic coma. The method we used to arrive at such an evaluation for each patient is described previously. Although this is a very qualitative method of evaluation, it seems to produce data that are of good clinical significance. Thus, 91% of all patients classified as being in "poor" clinical condition died. Seventy percent of those in the "fair" group died, but only 12% of those in the "good" category died. If a patient is classified as "poor" he will have little chance of survival.

If he is in "good" clinical condition he will probably survive. Over 70% of these 104 patients fell into these two groups. It is more difficult to prognosticate about the other patients, but it would seem that they have only a fair chance of surviving. (Figure IV)

The occurrence of hepatic coma in an individual patient is of ominous prognostic import. In fact, 94% of those patients who had hepatic coma at any time during their admission to the hospital died.

On the basis of the foregoing material a program may be evolved which would appear to offer the best chance of survival to patients bleeding from esophageal varices. This plan, in general, is in current usage at this institution. There is required full cooperation between a team of internists, esophagoscopists, radiologists and cardiovascular surgeons, made up of individuals who have carefully considered all of the aspects of this problem. This group acts in an advisory capacity meeting frequently, discussing individual patients, and supervising the house staff in the various phases of the management of the hemorrhage.

The prime step in the institution of a therapeutic program is an early and correct diagnosis. This can best be achieved by the prompt use of esophagoscopy by experienced personnel, prior to the use of esophageal tamponade. This is, by far, the best means of diagnosis. The use of BSP,

blood ammonium and other laboratory information are of value in those cases in whom esophagoscopy does not give a definitive answer.

If a diagnosis of bleeding from esophageal varices has been made the SBT should be passed immediately. This is a direct form of therapy, and it has been effective in controlling bleeding in most of our cases despite the fact that it has not, by itself, decreased overall mortality. It is certainly a temporary measure, but in view of the precarious situation of most of these patients, it should be used while other more definitive modes of therapy are being considered. The dangers inherent in the use of the SBT have been mentioned and constant attention to these factors will undoubtedly diminish the incidence of complications.

Those patients who are in good condition and whose bleeding has been controlled by the SBT should have one of the shunt procedures performed as soon as it is clinically feasible. Those patients who are in "poor" condition or who are in hepatic coma should not have a shunt until they have been treated medically and have improved. In these patients conservative means, including the SBT, are used. The strong argument against operation in these people is illustrated by our very poor results with transesophageal ligation. Almost all of our patients who underwent this operation were in poor clinical condition. The mortality of 87.5%

speaks for itself. In light of our experience transesophageal ligation plays no role in therapy.

The large group of patients who fall between those who are neither good nor poor risks are the most difficult to assess. They have suffered a rather high mortality (70%) under our past methods of treatment. It appears reasonable to treat these patients in the manner previously designated for those patients in the "good" category. Consequently these patients should be subjected to the shunt operations shortly after bleeding has been controlled. The criteria for operation in this group are based not on individual laboratory values, but on an overall clinical and laboratory impression.

The most difficult group to treat are those classified as "fair" but in whom bleeding has not been controlled. These may be treated by shunt procedures with the esophageal balloon in place, by continued esophageal tamponade alone, or by injection of varices. These cases must be treated individually according to the particular circumstances attending each. Obviously, the other adjuncts of general supportive therapy must be used with discrimination in all patients with bleeding esophageal varices. These include transfusions, antibiotics, sedation, vitamins, enemata and antacids.

Transcutaneous splenoportography is performed on the operating table pre-operatively to determine which type of

shunt can be performed most readily. The construction of portacaval shunts is preferred in patients with intra-hepatic obstruction, while splenorenal shunts are usually done in those with Banti's syndrome.

It is obvious that the most important factor in the recovery of these patients is their clinical status. In this series the high mortality rate seemed independent of the form of therapy employed. It is conceivable that no type of management will significantly alter these statistics. It is more likely, however, that periodic critical analyses of previous experience, such as this one, can emphasize the more effective methods of treatment. At the very least they can indicate in what directions it is too dangerous to trespass.

SUMMARY

The records of 104 patients who bled from esophageal varices were examined. The overall mortality was 72%. The most frequent cause of death was hemorrhage. Ninety-five patients were in the intra-hepatic group, and 9 were in the extra-hepatic group. The former group had a death rate of 75%, while only 43% of the extra-hepatic group died.

Seventy percent of 30 patients who were treated conservatively died. Hemorrhage accounted for 81% of these deaths.

The SBT controlled hemorrhage in 72% of 72 occasions. However, in the 50 patients in whom it was used there were 38 deaths (76%). Five patients died as a direct consequence of the use of the tube. Complications occurred in 38% of the occasions on which it was used.

The results in 13 patients who underwent portacaval shunts were generally good. Nine patients who underwent splenorenal shunts also had good results. These patients were in better condition than the patients treated medically.

Transesophageal ligation was carried out in 8 patients. Seven of these patients died. Almost all were in poor clinical condition.

A discussion of prognostic criteria is included and a program of management is outlined.

APPENDIX

Case Summaries

* indicates patients in whom esophageal varices were not proven.

1. B38342 I.A. was a 65 year old white man with metastatic cancer in his liver. He had a severe hematemesis. Bleeding was controlled with the SBT. A bleeding point in an esophageal varix was seen by esophagoscopy. He was discharged after 28 days. On the following day he had another hematemesis and was re-admitted to the hospital where the SBT again controlled the bleeding. Sclerosing injections were carried out on 4 varices. In spite of several tube complications and 2 episodes of shock the patient was discharged alive. He subsequently died of his underlying disease.

2. 40-14-93 F.B. was a 61 year old white man with cirrhosis. He had a severe hematemesis from esophageal varices which were demonstrated on X-ray. Bleeding was controlled with the SBT. He was discharged alive.

3. A22869 E.B. was a 55 year old white man with post-necrotic cirrhosis. He was admitted because of hepatic decompensation. X-rays showed esophageal varices. After 23 days of medical therapy for his cirrhosis he went into shock suddenly and died. Esophageal varices with massive hemorrhage was found at autopsy. No whole blood was given.

4. B94320 H.B. was a 48 year old white man with Laennec's cirrhosis admitted because of hematemesis. The SBT controlled the bleeding and the patient's condition stabilized nicely. On the 14th day a transesophageal ligation was carried out, but all varices were not ligated. He died 7 days later of recurrent bleeding and hepatic coma.

5. 41-83-55 L.B. was a 52 year old white woman with Laennec's cirrhosis who had a severe hemorrhage from esophageal varices demonstrated on X-ray. The SBT controlled the bleeding. On the 10th day she underwent a portacaval shunt. She had a benign recovery. A year later she was alive and well.

6. C54235 H.B. was a 60 year old white woman who had a severe hemorrhage from esophageal varices. A laparotomy showed the bleeding to be coming from above the stomach and a Rowntree tube was passed. This did not control the bleeding and the patient died a day later. Portal vein thrombosis and ruptured esophageal varices were seen at autopsy.

7. A60734 E.B. was a 64 year old white man with Laennec's cirrhosis who had a severe hematemesis. An attempt was made to control the bleeding with a Miller-Abbot tube which was inflated and pulled up against the cardia. This did not control the bleeding and the patient died on the 3rd day. Ruptured varices were seen at autopsy.

8. B36858 W.B. was a 38 year old white man with Laennec's cirrhosis. He had a massive hemorrhage from esophageal varices which were seen on X-ray. Treatment consisted of whole blood and supportive therapy. He was discharged alive after 25 days.

9. A61985 S.B. was a 65 year old white man admitted because of precordial pain and shortness of breath. He was treated as a case of myocardial infarction and died after a few hours. At autopsy post-necrotic cirrhosis and massive hemorrhage from esophageal varices were demonstrated. He received no specific therapy.

10. C9133 J.B. was a 62 year old white man with Laennec's cirrhosis. He was admitted because of hepatic decompensation. Within a few hours of admission to the hospital he suddenly vomited blood, aspirated a good deal if it, and died. Bleeding esophageal varices were seen at autopsy.

11. 38-60-88 E.B. was a 32 year old white woman with Banti's disease. The SBT controlled an episode of severe bleeding from varices which had been seen during esophagoscopy. However, there were several tube complications. A splenectomy was done on the 11th day. Fifteen months later she had a second hematemesis. The SBT did not control the bleeding. An esophago-gastrectomy was done. She died 3 days afterward, of recurrent hemorrhage and hepatic coma.

* 12. B61472 S.B. was an 18 year old white girl with post-necrotic cirrhosis admitted for hematemesis. A GI series was negative. She developed an E.Coli septicemia and died in 3 days of recurrent bleeding and hepatic coma. She received only supportive therapy.

13. B96343 L.B. was a 61 year old white man with Laennec's cirrhosis admitted for cardiac and hepatic decompensation. His stools contained occult blood and he was transfused. After several weeks he began vomiting blood and despite repeated transfusions and conservative medical therapy he died after a month and a half. Varices were seen on X-ray.

14. 44-28-12 A.B. was a 42 year old white woman who had a hematemesis while in the hospital following a cholycystectomy. The SBT controlled the bleeding from varices which had been demonstrated on X-ray, but she aspirated vomitus from around the tube. She died and hemochromatosis with ruptured varices was found at autopsy.

15. A22286 S.C. was a 51 year old white man with Laennec's cirrhosis admitted because of tarry stools. Esophagoscopy demonstrated esophageal varices. He was treated conservatively and after 45 days a splenorenal shunt was constructed. He was alive and well 3 years later and no varices could be seen on esophagoscopy.

16. C25661 D.C. was a 49 year old Negro man with Laennec's cirrhosis. He was admitted because of hepatic decompensation. His stools were guiac positive and he was thought to be oozing slowly from esophageal varices demonstrated on GI series. He was transfused and his hemoglobin rose from 9.5 to 13. About 3 months later he vomited blood. In spite of transfusions and conservative therapy he lapsed into coma and died after a slow downhill course.

17. C34832 F.C. was a 65 year old white man with Laennec's cirrhosis who was admitted for hematemesis. In spite of multiple transfusions and the use of a crude balloon tube he lapsed into coma and died within 2 days. Ruptured esophageal varices were seen at autopsy.

18. 36-81-95 K.C. was a 49 year old white woman with Laennec's cirrhosis who was admitted in hepatic decompensation. Upon admission she vomited blood. The SBT controlled the bleeding but there were several tube complications and she died on the 15th day in hepatic coma. Ruptured varices were seen at autopsy.

19. C54774 M.C. was a 62 year old white man with Laennec's cirrhosis. He had a severe hemorrhage from esophageal varices which were seen on X-ray. The bleeding stabilized with conservative therapy and he was discharged after a month. Five months later he vomited blood. A splenic and hepatic arterial ligation was carried out. The patient died in hepatic coma.

20. C38627 D.D. was a 65 year old white man with Laennec's cirrhosis admitted because of tarry stools. He was treated with the Rowntree tube and transfusions but he died of recurrent bleeding.

21. 44-96-54 L.D. was a 50 year old white woman with Laennec's cirrhosis who had been treated at another hospital for bleeding varices. She was transferred to this hospital. Bleeding was controlled with the SBT and despite early hepatic coma a transesophageal ligation was done. She developed an empyema. On the 14th day post-operatively she vomited blood. The SBT was again successful in controlling the hemorrhage, but she went into coma and died shortly thereafter.

22. B60639 S.D. was a 49 year old white man with Laennec's cirrhosis who was admitted for hematemesis. Esophagoscopy had shown varices. The SBT was successful in controlling bleeding. After 2 weeks a porta-caval shunt was constructed. The patient was alive a year later.

* 23. B95665 D.D. was a 29 year old white woman with biliary cirrhosis who vomited blood. She was discharged after a 3 week course of transfusions and conservative therapy. Five months later she again vomited blood and despite this type of treatment she died in hepatic coma on the 4th day. Varices were never demonstrated, but the clinical impression was that her bleeding had come from them.

24. 37-67-60 A.D. was a 55 year old white woman with Laennec's cirrhosis who had had a splenectomy 1 month prior to admission. She vomited blood. An SBT controlled bleeding despite several complications and she was discharged alive on the 43rd day.

25. 46-55-48 W.F. was a 49 year old white man with Laennec's cirrhosis. While in the hospital for decompensated cirrhosis occult blood was found in his stools. He died the following day. The pathologist attributed the bleeding to esophageal varices found at autopsy.

26. B96543 W.F. was a white man in his fifties who was admitted on 4 occasions for bleeding from esophageal varices. He had Banti's disease. He underwent splenectomy. He died 4 years after his first hemorrhage from recurrent bleeding.

27. C47250 P.G. was a 72 year old white woman with Laennec's cirrhosis who vomited blood while in the hospital for hepatic failure. Esophageal varices were seen on X-ray. She received whole blood and was discharged alive 2 weeks later.

28. A23137 A.G. was a 68 year old white man admitted for decompensated Laennec's cirrhosis. On the 11th day he suddenly vomited blood and died. Ruptured esophageal varices were found at autopsy.

29. C36483 A.G. was a 63 year old white man with cirrhosis. He vomited blood and was treated for 4 days with bed rest and transfusions. Bleeding seemed to have stopped, when he went into shock and died. Esophageal varices were demonstrated at autopsy.

30. 35565 I.G. was a 55 year old white woman with post-necrotic cirrhosis who was admitted for hematemesis. The SBT was not successful in controlling the bleeding and there were several complications due to its use. She died after 8 days. Ruptured esophageal varices were seen at autopsy.

31. 39-19-63 P.G. was a 44 year old white man with Laennec's cirrhosis who was admitted from another hospital where he had been treated for bleeding esophageal varices with only fair success. He was transfused, and 2 weeks later a portacaval shunt was constructed. Four months later he was alive and well.

* 32. 36747 L.G. was a 57 year old white woman with Laennec's cirrhosis who was admitted in hepatic coma. On the 9th day she vomited blood. Although the SBT controlled the bleeding it was directly responsible for her death due to aspiration.

33. B18783 J.G. was a 61 year old white man with Laennec's cirrhosis and superimposed hepatoma. He passed persisting guiac positive stools while in the hospital and finally died a month after admission. He was treated conservatively. Esophageal varices were seen at autopsy.

34. 43-40-26 A.H. was an 82 year old white woman with biliary cirrhosis who was admitted for liver decompensation. While in the hospital she vomited blood, was treated conservatively and died. Ruptured esophageal varices were seen at autopsy.

35. A20880 J.H. was a 69 year old white man with Laennec's cirrhosis who was admitted with a history of having passed tarry stools. Esophageal varices were seen to be actively bleeding on esophagoscopy. A portacaval shunt was done after multiple transfusions and the patient was discharged alive.

36. 45-65-12 E.H. wasa 63 year old white woman with post-necrotic cirrhosis who was admitted because of hematemesis. Esophagoscopy showed varices. On the 18th day a portacaval shunt was constructed. Aside from a paranoid psychosis post-operatively, the patient did well.

37. A67585 B.H. was a 38 year old white woman with biliary cirrhosis admitted for vomiting blood. The SBT did not control the bleeding. A transesophageal ligation was carried out on the 3rd day. The following day she went into hepatic coma and died. No post-operative bleeding was demonstrated at autopsy.

38. 99894 C.H. was a 74 year old white man with post-necrotic cirrhosis admitted because of tarry stools. The SBT controlled bleeding. Esophageal varices were seen at esophagoscopy and a transesophageal ligation was carried out on the 3rd day. He died the next day of recurrent hemorrhage.

39. B80405 B.J. was a 74 year old Negro woman with Laennec's cirrhosis admitted because of hematemesis. Despite complications, the SBT controlled the bleeding. However several episodes of respiratory obstruction were probably directly due to the SBT and she died rather suddenly. Ruptured esophageal varices were seen at autopsy.

* 40. C55490 W.K. was a 38 year old white man with Laennec's cirrhosis admitted because of vomiting blood. A Rowntree tube was not successful in controlling the bleeding and he died on the 3rd day. A barium swallow could not prove the presence of esophageal varices, but it was the clinical impression that this was the source of his bleeding.

41. 45-41-50 M.K. was a 61 year old white man with Laennec's cirrhosis. His bleeding was shown at esophagoscopy to be coming from ruptured esophageal varices. A portacaval shunt was immediately done. He passed into hepatic coma and died 4 days later.

* 42. C28124 R.L. was a 57 year old white man with Laennec's cirrhosis admitted because of hematemesis. Esophageal varices were never demonstrated conclusively. However this diagnosis was made on clinical grounds. He had never had ulcer symptoms. Despite conservative therapy he died of hemorrhage with aspiration of vomitus on the day of admission.

43. 45-63-02 G.M. was a 53 year old white man with Laennec's cirrhosis who was transferred here from another hospital with poorly controlled bleeding from esophageal varices. The SBT did not control bleeding and there were several complications consequent to its use. He continued to bleed and went into hepatic coma before his death. Ruptured varices were seen at autopsy.

44. B17101 W.M. was a 64 year old white man with Laennec's cirrhosis admitted because of hematemesis. The SBT failed to control bleeding and he died 31 hours after admission. Autopsy revealed esophageal varices as the probable site of bleeding.

45. 38230 O.O. was a 79 year old white man with biliary cirrhosis admitted because of hematemesis. He was treated conservatively and received sclerosing injections 2 weeks after the acute bleeding. He was discharged after 18 days but died within a year at his home following a massive hematemesis.

46. B11702 G.O. was a 65 year old white man with Laennec's cirrhosis admitted for hematemesis. The SBT was successful in controlling bleeding. An exploratory laparotomy was performed and anatomical conditions were found that would have prevented the establishment of a portacaval shunt. He developed hepatic coma after this, had another hemorrhage and died. Ruptured varices were seen at autopsy.

47. A88833 V.P. was a 66 year old white man with Laennec's cirrhosis who was admitted because of hematemesis. He was treated conservatively and died within 4 hours. Esophagoscopy was negative but ruptured esophageal varices were seen at autopsy.

48. 39-70-90 H.P. was a 56 year old white man with Laennec's cirrhosis admitted for hematemesis. The SBT was successful in controlling the bleeding and was removed. However bleeding commenced again and this time the SBT did not control the bleeding. On the 7th day he went into shock and died. Ruptured varices were seen at autopsy.

49. B67507 M.P. was a 37 year old white woman with hemochromatosis admitted for hematemesis. The SBT was passed with difficulty and seemed to control bleeding. Esophagoscopy was performed on the 8th day and three varices were injected. At this time the esophagus was perforated and she developed an empyema and tension pneumothorax. She died in hepatic coma probably as a consequence of her infection.

50. 38-34-67 C.R. was a 67 year old white woman with cirrhosis of unknown etiology admitted for hematemesis. Esophagoscopy showed esophageal varices. The SBT controlled bleeding. She then underwent a cholycystostomy. She continued to hemorrhage and the SBT was again used successfully. A splenorenal shunt was constructed, but she died soon afterwards of pneumonia.

51. 44-43-97 D.R. was a 53 year old white woman with post-necrotic cirrhosis admitted for probable subacute necrosis of the liver secondary to hepatitis. She vomited blood on the 4th day and the SBT failed to control bleeding. The tube also caused respiratory embarrassment. She died 2 days later and ruptured varices were seen at autopsy.

52. 38-30-06 M.R. was a 57 year old white man with Laennec's cirrhosis admitted for massive hematemesis. The SBT controlled the bleeding but 20 hours after admission he vomited material around the tube, aspirated much of it and died. Ruptured varices were seen at autopsy.

53. C57741 W.R. was a 56 year old white woman with post-necrotic cirrhosis admitted for hematemesis. Despite several complications the SBT controlled the bleeding. The patient died on the 8th day as a direct consequence of the use of the SBT. Esophageal varices were seen at autopsy.

54. 45-96-98 M.R. was a 54 year old white man with Laennec's cirrhosis admitted for hematemesis. Esophagoscopy demonstrated bleeding esophageal varices. A trans-esophageal ligation was carried out 39 hours after admission. The patient developed hepatic coma. Seven days later he was found to have developed an esophago-pleuro-cutaneous fistula. He was treated with antibiotics. A week later he died of respiratory failure.

55. B37018 H.R. was a 69 year old white woman with primary carcinoma of the liver. Despite conservative therapy she died of massive hemorrhage and hepatic coma 3 days after admission. Ruptured esophageal varices were found at autopsy.

56. A47621 M.S. was a 35 year old white woman with Laennec's cirrhosis admitted for hematemesis. She was treated conservatively but died 28 hours after admission of recurrent bleeding. Autopsy revealed ruptured esophageal varices.

57. 37-18-59 F.S. was a 77 year old white woman with Laennec's cirrhosis admitted because of melena and hematemesis. Despite conservative treatment she died on the 3rd day in pulmonary edema. Ruptured esophageal varices were seen at autopsy.

58. 85873 O.S. was a 33 year old Negro woman with Laennec's cirrhosis admitted in coma. Esophageal varices had been seen on X-ray during a previous admission. Two hours after admission she vomited blood. An attempt was made to pass the SBT without success. She died within 2 hours of this. She was given whole blood and anti-shock measures.

59. B51553 A.S. was a 65 year old white man with Laennec's cirrhosis and superimposed hepatoma. While in the hospital for his underlying disease he vomited blood. Despite transfusions he died of his hemorrhage. Ruptured esophageal varices were seen at autopsy.

60. 41-84-89 S.S. was a 23 year old white man with Banti's disease admitted for hematemesis. The SBT did not control the bleeding. On the 2nd day a transesophageal ligation was carried out. Bleeding was noted to be mainly from gastric varices. Twenty-three days later a splenorenal shunt was constructed. Six months later the patient was found to be oozing slowly from his varices. Injection therapy was given. He is alive 3 years after his first episode.

61. 37-07-64 A.S. was a 46 year old white man with Laennec's cirrhosis admitted because of melena. The SBT controlled the bleeding despite several complications. He died in coma on the 17th day. Ruptured varices were found at autopsy.

62. B95115 J.S. was a 77 year old white man with Laennec's cirrhosis admitted for hematemesis. He was treated with transfusions and injection of the varices. After 2 weeks he was discharged. A year later he died of cancer of the stomach.

63. C42489 G.S. was a 56 year old Negro woman with Laennec's cirrhosis admitted for hematemesis. The SBT controlled the bleeding but caused her death, which was due to aspiration. Ruptured esophageal varices were found at autopsy.

* 64. C28204 M.S. was a 37 year old white woman with Laennec's cirrhosis admitted because of melena and blood in her stools. She began to vomit bright red blood. Esophageal varices were not demonstrated but this was felt to be the site of her bleeding. Despite conservative measures and a Rowntree tube she died of hemorrhage and hepatic coma.

65. A86668 W.T. was a 65 year old white man with Laennec's cirrhosis admitted for hematemesis. Esophageal varices were seen on X-ray. He was treated conservatively and survived. A year later he survived another similar episode. Finally, a month after this latter episode he vomited blood, was treated with transfusions and died of massive hemorrhage within 24 hours.

66. 44-89-80 D.W. was a 42 year old Negro woman with Laennec's cirrhosis admitted for hematemesis. Despite conservative measures and transfusions she died on the 5th day. Esophageal varices were found at autopsy.

67. 43-90-31 C.W. was a 72 year old white man with post-necrotic cirrhosis admitted for hematemesis. The SBT failed to control bleeding. A portacaval shunt was done on the 9th day. The patient continued to bleed despite a second SBT, went into coma and died. Autopsy revealed esophageal varices.

* 68. 57603 W.W. was a 77 year old white woman with biliary cirrhosis admitted for hematemesis. The SBT failed to control bleeding. Esophageal varices were not demonstrated, but were felt to be the source of the bleeding. She died on the 2nd day in pulmonary edema.

69. 41-11-52 L.W. was a 37 year old white man with sarcoidosis. An episode of hematemesis caused him to come to the hospital. The SBT controlled the bleeding. A portacaval shunt was done on the 9th day. Despite a post-operative subhepatic abscess he recovered and was alive a year later. X-rays had shown esophageal varices.

70. 43-71-55 L.W. was a 45 year old white woman with sarcoidosis. She was admitted for evaluation. X-rays showed esophageal varices. On the 6th day of hospitalization she passed a large tarry stool. She was treated with transfusions and discharged 9 days later.

71. C30460 C.Z. was a 56 year old white man with Laennec's cirrhosis admitted for coma. He was found to be oozing slowly from esophageal varices and was treated conservatively. Seven months later he began bleeding rectally. The SBT controlled the bleeding and on the 29th day a portacaval shunt was attempted. He died on the operating table despite intra-arterial transfusions. Autopsy revealed esophageal varices.

72. B22960 O.A. was a 65 year old white woman with biliary cirrhosis. While on the Orthopedic Service she vomited blood. GI series showed esophageal varices. The SBT was unsuccessful. She died after a month of conservative therapy of recurrent hemorrhage.

* 73. 43-68-63 H.B. was a 55 year old white woman with Laennec's cirrhosis admitted for hematemesis. The SBT controlled the bleeding. However, she lapsed into hepatic coma and died on the third day. Esophageal varices were never demonstrated, but it was the clinical impression that she was bleeding from them.

* 74. A91468 N.M. was a 62 year old white man with Laennec's cirrhosis admitted for hematemesis. The SBT controlled bleeding despite complications, but the patient died of hepatic coma on the 5th day. It was the clinical impression that this man bled from esophageal varices.

* 75. 60691 A.V. was a 65 year old white woman with Laennec's cirrhosis admitted for hematemesis. The SBT failed to control bleeding and the patient bled to death. During her many previous admissions for hepatic decompensation GI series had been done and no varices were demonstrated. However, she had no lesions in the stomach or duodenum either.

76. 38-69-72 G.K. was a 60 year old white man with Laennec's cirrhosis admitted for tarry stools. He was treated with whole blood and discharged a month later. The impression was bleeding from esophageal varices. A year later he was admitted in coma and was not given any transfusions. He died within 2 days. Autopsy revealed ruptured esophageal varices.

77. 81984 H.M. is a young white man with portal vein thrombosis. He has survived over 20 episodes of bleeding from esophageal varices and has had various methods of treatment including conservative therapy, injection therapy, a splenectomy, and a spleno-renal shunt with venous graft. He is alive 20 years after his first episode.

78. 38-20-24 J.A. was a 33 year old white man with cirrhosis of the liver. He was admitted for hematemesis, was treated conservatively and was discharged alive. Esophageal varices were noted on X-ray.

79. 40-11-26 G.B. was a 46 year old white woman with biliary cirrhosis admitted for hepatic decompensation. GI series showed esophageal varices. While in the hospital she passed tarry stools. The SBT was passed, she was taken to the operating room and a transesophageal ligation was done. She developed hepatic coma thereafter. Sixteen days post-operatively she again had tarry stools. The SBT was passed and she went into cardiac arrest. She recovered and then underwent a splenorenal shunt. She died a few days later of hemorrhage and hepatic coma.

80. C49273 K.B. was a 42 year old white woman with Laennec's cirrhosis admitted for hematemesis. The SBT controlled bleeding and she was discharged 9 days later. A year later she underwent an elective splenorenal shunt. Five years later she vomited blood, went into hepatic coma and died within 3 days. At autopsy ruptured varices and an obliterated splenorenal shunt were found.

81. 92760 G.D. was a 62 year old white woman with Laennec's cirrhosis admitted for hematemesis. Esophagoscopy revealed bleeding varices. The SBT was passed and later that day she underwent a transesophageal ligation. She lapsed into coma and died a few days later.

82. C48486 S.D. was a 52 year old white man with cirrhosis of unknown etiology admitted for hematemesis. Esophageal varices were seen on X-ray. The SBT controlled the bleeding. After a few weeks he underwent splenic and hepatic arterial ligation. He was discharged alive.

83. 42-62-71 E.D. was a 62 year old white man with sarcoidosis of the liver admitted because of hematemesis. A portacaval shunt was constructed 4 days after admission. Esophageal varices had been seen on X-ray. He was discharged and was alive and well 2 months later.

84. 37-06-73 E.E. was a 47 year old white woman with Laennec's cirrhosis admitted in hepatic coma. Stools were guiac positive. An attempt was made to pass the SBT but the patient immediately coughed it up and went into respiratory failure. She died. Autopsy revealed ruptured esophageal varices.

85. 37-76-50 G.L. was a 27 year old white woman with cirrhosis of the liver. She was admitted in hepatic decompensation and on the 11th day passed tarry stools. Despite conservative therapy she died a month later. Autopsy revealed ruptured esophageal varices.

* 86. 38-57-52 G.S. was a 65 year old white man with Laennec's cirrhosis admitted for hematemesis. He was treated conservatively and discharged alive after 2 weeks. GI series was indeterminate for varices. However, the clinical impression was bleeding from esophageal varices.

87. 46-38-11 C.S. was a 51 year old white man with Banti's disease admitted because of vomiting blood. A portacaval shunt was constructed and he made a good recovery. GI series had shown esophageal varices.

88. C62897 C.M. was a 33 year old Puerto Rican woman with schistosomiasis. Although the SBT did not control her bleeding she survived and underwent an esophagogastrectomy and a splenorenal shunt. She is alive after 6 years.

89. 39-24-27 M.B. was a 27 year old white woman with post-necrotic cirrhosis who was admitted because of decompensation. While in the hospital she vomited blood. The SBT controlled the bleeding but she died in hepatic coma 3 days later. Autopsy revealed what were probably esophageal varices.

90. C50367 R.B. was a 26 year old white man with Banti's disease admitted because of hematemesis. A splenorenal shunt was performed after 3 weeks of conservative therapy and he was alive 7 years later.

91. 40-71-26 S.D. was a 58 year old white man with Laennec's cirrhosis admitted because of hepatic cecompensation. GI series revealed esophageal varices. After a month of hospitalization he was felt to be slowly oozing from his varices. He had one bout of hematemesis. Despite transfusions he died in hepatic coma, probably of hemorrhage and aspiration.

92. 47-50-26 E.F. was a 41 year old white woman with post-necrotic cirrhosis admitted for hematemesis. GI series showed esophageal varices. A week later, after conservative therapy, she underwent a portacaval shunt. She recovered and was alive 3 months later.

93. 47-78-41 A.H. was a 58 year old white woman with Laennec's cirrhosis admitted for ascites. After a month of hospitalization she vomited blood. Esophagoscopy revealed bleeding varices. The SBT controlled the bleeding and was removed after 48 hours. She began to bleed again 4 days later and the tube couldn't be passed. She aspirated vomitus and died.

94. 47-98-60 M.J. was a 75 year old white man with Laennec's cirrhosis admitted for hematemesis. Esophagoscopy revealed bleeding varices. The SBT controlled the bleeding and was removed. The patient began to bleed again and this time the SBT did not control the bleeding. The patient died 9 days after the onset.

95. 47-91-21 L.R. was a 49 year old white woman with Laennec's cirrhosis admitted in coma. A gastric tube was passed and blood was aspirated from the stomach. She died the following day. Autopsy revealed ruptured esophageal varices.

96. 47-31-24 V.T. was a 74 year old white man with Laennec's cirrhosis admitted because of hematemesis. GI series revealed esophageal varices. Despite conservative therapy he died the next day.

97. 47-00-68 V.W. was a 50 year old white woman with Laennec's cirrhosis admitted for hematemesis. The SBT controlled bleeding but the patient went into pulmonary edema and died after 4 days. Autopsy revealed ruptured esophageal varices.

98. B68072 C.J. was a 43 year old Negro man with cirrhosis who was admitted for hematemesis. He died of hemorrhage and aspiration despite conservative measures after 3 days. Autopsy revealed ruptured esophageal varices.

99. B78803 H.K. was a 62 year old white man with Laennec's cirrhosis admitted for hematemesis. Esophageal varices were seen on X-ray. He died of hemorrhage and hepatic coma a few days later despite conservative therapy.

100. A15392 J.M. was a 36 year old white man with Laennec's cirrhosis admitted for hematemesis. GI series showed esophageal varices. He was treated conservatively and discharged alive after 3 weeks. He was alive 3 years later.

101. B51553 A.S. was a 65 year old white man with Laennec's cirrhosis and superimposed carcinoma of the liver admitted for hematemesis. He died within 7 hours despite transfusions. Esophageal varices were seen at autopsy.

102. A98645 W.R. was a 58 year old white man with Laennec's cirrhosis admitted for hematemesis. Esophagoscopy revealed esophageal varices. The SBT controlled the bleeding. A month later he had a portacaval shunt. He had demonstrated an ability to survive an episode of hepatic coma.

103. 48-17-64 R.T. was a 34 year old white man with Laennec's cirrhosis admitted for tarry schools. Esophagsocopy revealed esophageal varices. The SBT did not control the bleeding. Five days after admission he underwent a portacaval shunt. He did well post-operatively.

104. 48-14-88 H.K. was a 48 year old white woman with post-necrotic cirrhosis. She had had 3 episodes of hematemesis and survived all of them. Esophagoscopy revealed esophageal varices. She underwent an elective splenorenal shunt here and was discharged alive.

BIBLIOGRAPHY

1. Barborka, C.J., and Texter, E.C.: Peptic Ulcer and Benign Gastric Lesions, in Christopher's Textbook of Surgery, 6th ed. Philadelphia, W.B. Saunders Company, 1956.

2. Barg, E.H., and Dulin, J.W.: Splenectomy in Treatment of Banti's Syndrome, A.M.A. Arch. Surg. 41:91, 1940.

3. Baronofsky, I.D.: Portal Hypertension: with Special Reference to the Acid Peptic Factor in the Causation of Hemorrhage and Extensive Gastric Resection in its Treatment. Surg. 25:135, 1949.

4. Berg, A.A., and Rosenthal, N.: Ligation of Splenic Artery, J. Mt. Sinai Hosp. 8:382, 1942.

5. Berman, J.K., and Hull, J.E.: Hepatic, Splenic and Left Gastric Arterial Ligations in Advanced Portal Cirrhosis, A.M.A. Arch. Surg. 65:37, 1952.

6. Blain, A.W., and Blain, A.: Ligation of the Splenic Artery, Operation of Choice in Selected Cases of Portal Hypertension and in Banti's Syndrome, Ann. Surg. 131:92, 1950.

7. Blakemore, A.H.: Portacaval Shunt for Portal Hypertension, J.A.M.A. 145:1335, 1951.

8. Blakemore, A.H.: Portacaval shunting for portal hypertension. Surg. Gyn. Obst. 94:443, 1952.

9. Blakemore, A.H.: Treatment of Bleeding Esophageal Varices with Balloon Tamponage, N.Y. State J. Med. 54:2057, 1954.

10. Carlgren, L.E., Lichtenstein, A., and Sandblom, O.: A Contribution to the Knowledge of Banti's Syndrome, Acta Paediat. Suppl. 75, 1949.

11. Child, C.G.: The Portal Circulation. New Eng. J. Med. 252:837, 1955.

12. Conn, H.O.: Hazards Attending the use of Esophageal Tamponade. To be published.

13. Crafoord, C. and Frenckner, P.: New Surgical Treatment of Varicose Veins of the Oesophagus, Acta Otol. 27:422, 1939.

14. Crile, G.: Transesophageal Ligation of Bleeding Esophageal Varices, A.M.A. Arch. Surg. 61:654, 1950.

15. Crile, G.: Treatment of Esophageal Varices by Transesophageal Obliteration, Surg. Gyn. Obst., 96:573, 1953.

16. Crohn, B. in Panel Meeting on Therapeutic Management of Severe Upper Gastrointestinal Hemorrhage, Bull. N.Y. Acad. Med. 33:405, 1957.

17. Douglass, B.E., and Snell, A.M.: Portal Cirrhosis: An Analysis of 444 cases with notes on Modern Methods of Treatment. Gastroenterology 15:407, 1950.

18. Ellis, D.S., Linton, R.R., and Jones, C.M.: Effect of Venous-Shunt Surgery on Liver Function in Patients with Portal Hypertension, New Eng. J. Med. 254:913, 1956.

19. Ekman, C.A.: Portal Hypertension, Acta Chir. Scand., Suppl. 222, 1957.

20. Everson, T.C., and Cole, W.H.: Ligation of the Splenic Artery in Patients with Portal Hypertension, A.M.A. Arch. Surg. 56:153, 1948.

21. Fainer, D.C., and Halsted, J.A.: Sources of Upper Alimentary Tract Hemorrhage in Cirrhosis of the Liver, J.A.M.A. 157:413, 1955.

22. Friedman, E., Katz, D., and Selesnick, S.: Flexible Esophagoscopy, N.Y. State J. Med. 58:351, 1958.

23. Garlock, J.H. and Som, M.L.: Further Observations on Packing of Mediastinum for Esophageal Varices, J. Thorac. Surg. 19:572, 1950.

24. Hallenbeck, G.A., and Shockett, E.: An Evaluation of Portacaval Shunts for Portal Hypertension, Surg. Gyn. Obst. 105:49, 1957.

25. Hamilton, J.E.: The Management of Bleeding Esophageal Varices Associated with Cirrhosis of the Liver, Ann. Surg. 141:637, 1955.

26. Higgins, E.H.: The Esophageal Varix. A Report of 115 Cases, Am. J. Med. Sci. 214:436, 1947.

27. Howells, L.: The Treatment of Splenic Anemia and Banti's Syndrome, Lancet 234:1320, 1938.

28. Hunt, A.H.: Portal Hypertension. Edinburgh and London, E. and S. Livingstone, Ltd., 1958.

29. Jahnke, E.J., Hughes, C.W., and Campbell, D.: Portal Hypertension. U.S. Armed Forces Med. J. 5:21, 1954.

30. Julian, O.C., and Dye, W.S.: Venous Shunts in Portal Hypertension, A.M.A. Arch. Surg. 63:373, 1951.

31. Large, A., Johnston, C.G., and Preshaw, D.E.: The Portacaval Venous Shunt, Ann. Surg. 135:22, 1952.

32. Linton, R.R.: Emergency Treatment of Bleeding Esophageal Varices and Results of Portacaval Shunts in 90 patients, N.Y. State J. Med. 53:2192, 1953.

33. Linton, R.R., and Ellis, D.S.: Emergency and Definitive Treatment of Bleeding Esophageal Varices, J.A.M.A. 160:1017, 1956.

34. Linton, R.R., and Warren, R.: The Emergency Treatment of Massive Bleeding from Esophageal Varices by Transesophageal Suture of These Vessels at the time of Acute Hemorrhage, Surg. 33:243, 1953.

35. Lyons, C., and Patton, T.B.: Editorial on Bleeding Esophageal Varices, Surg. 39:540, 1956.

36. Madden, J.L.: Ligation of the Hepatic and Splenic Arteries in the Treatment of Cirrhosis of the Liver, Surg. Gyn. Obst. 96:594, 1953.

37. Macbeth, R.: Treatment of Oesophageal Varices in Portal Hypertension by means of Sclerosing Injections, Brit. Med. J. 2:877, 1955.

38. McDermott, W.V.: A Simple Discriminatory Test for Upper Gastrointestinal Hemorrhage, New Eng. J. Med. 257:1161, 1957.

39. McDermott, W.V., Wareham, J., and Riddell, A.G.: Bleeding Esophageal Varices. A Study of the Cause of Associated Hepatic Coma, Ann. Surg. 144:318, 1956.

40. McFadzean, A.J.S., and Cook, J.: Ligation of the Splenic and Hepatic Arteries in Portal Hypertension, Lancet 264:615, 1953.

41. Moersch, H.J.: Treatment of Esophageal Varices by Injection of a Sclerosing Solution, J.A.M.A. 135:754, 1947.

41A. Morgagni, J.B.: De Sedibus et causis morborum per anatomen indigatis T. III, Neapoli 1762, p. 126.

42. Nachlas, M.M.: Experiences with the Triple-Lumen Single-Balloon Tube in Massive Upper Gastrointestinal Hemorrhage. Gastroenterology 60:913, 1956.

43. Nachlas, M.M., O'Neil, J.E., and Campbell, A.J.A.: The Life History of Patients with Cirrhosis of the Liver and Bleeding Esophageal Varices, Ann. Surg. 141:10, 1955.

44. O'Sullivan, W.D., and Payne, M.A.: The Emergency Portacaval Shunt, Surg. Gyn. Obst. 102:668, 1956.

45. Palmer, E.D.: Chronic (Non-cirrhotic) Diffuse Liver Disease, Am. J. Digest. Dis. 1:499, 1956.

46. Paltis, V., and Sulamaa, M.: On the Surgical Treatment of Banti's Syndrome, Acta Chir. Scand. 109:106, 1955.

47. Patek, A.J., Post, J., Ratnoff, O.D., Mankin, H., and Hillman, R.W.: Dietary Treatment of Cirrhosis of the Liver, J.A.M.A. 138:543, 1948.

48. Patterson, C.O., and Rouse, M.O.: The Injection Treatment of Esophageal Varices, J.A.M.A. 130:384, 1946.

49. Phemister, D.B., and Humphreys, E.M.: Gastro-Esophageal Resection and Total Gastrectomy in the Treatment of Bleeding Varicose Veins in Banti's Syndrome, Ann. Surg. 126:397, 1947.

49A. Raciborski, A.: Histoire de decouvertes relatives au systeme veineux, envisage sans le rapport anatomique, physiologique, pathologique et therapeutique, depuis Morgagni jusqu'a nos jours. Mem. Acad. Roy. de Med. 9:447, 1841.

49B. Raikem, A.F.J.: Observations, reflexions et apercus sur quelques affections morbides de la veine porte, et notament sur l'inflammation, l'ossification de ce tronc vasculaire. Mem. Acad. Roy. de Med de Belgique 1:38, 1848.

50. Ratnoff, O.D., and Patek, A.J.: The Natural History of Laennec's Cirrhosis of the Liver. An Analysis of 386 Cases, Medicine 21:207, 1942.

51. Reynolds, T.B., Freedman, T., and Winsor, W.: Results of the Treatment of Bleeding Esophageal Varices with Balloon Tamponage, Am. J. Med. Sci. 224:500, 1952.

52. Rienhoff, W.F., and Woods, A.C.: Ligation of Hepatic and Splenic Arteries in Treatment of Cirrhosis with Ascites, J.A.M.A. 152:687, 1953.

53. Ripstein, C.B.: Experiences with Portacaval Anastomosis in the Treatment of Portal Hypertension, Surg. 34:570, 1953.

53A. Rokitansky, C.: Drey merkwurdige Falle von Erkrankungen des Pharynx und Oesophagus. Med. Jahrbucher des K.K. Osterr. Staates 21:230, 1840.

54. Rousselot, L.M.: Combined (One-stage) Splenectomy and Portacaval Shunts in Portal Hypertension, J.A.M.A. 140:282, 1949.

55. Rousselot, L.M. in Panel Meeting on Therapeutic Management of Severe Upper Gastrointestinal Hemorrhage, Bull. N.Y. Acad. Med. 33:405, 1957.

55A. Rowntree, L.G., Zimmermann, E.F., Todd, M.H. and Ajac, J.: Intraesophageal venous tamponage; itsusein a case of variceal hemorrhage from the esophagus, J.A.M.A. 135:630, 1947.

56. Santy, P., and Marion, P.: Obstructions portales et maladie de Banti, Maroc Medical 33:367, 1954.

57. Sengstaken, R.W., and Blakemore, A.H.: Balloon Tamponage for the Control of Hemorrhage from Esophageal Varices, Ann. Surg. 131:781, 1950.

58. Senn, A., and Blakemore, A.H.: Neun Johre Oesophagusbehandlung durch portale Dekompression. Erfahrungen und Ergebnisse. Chirurg. 26:217, 1955.

59. Schafer, P.W., and Kittle, C.F.: Partial Esophago-Gastrectomy in the Treatment of Esophagogastric Varices, A.M.A. Arch. Surg. 61:235, 1950.

59A. Vesalius, A.: De humani corporis fabrica. Basileae, 1555, p. 66.

60. Walker, R.M.: Portacaval Anastomosis, Lancet. Jan. 12, 1957.

61. Walker, R.M.: The place of venous shunts in the treatment of portal hypertension, Ann. Roy. Coll. Surg. England 14:145, 1954.

62. Watson, R.B.: Ligation of splenic artery for advanced splenic anemia, Brit. Med. J. 1:821, 1935.

63. Weber, J.M., Nash, E.C., and Gregg, L.A.: Hemorrhage from the Upper Gastrointestinal Tract, J.A.M.A. 165: 1899, 1957.

64. Welch, C.S.: Ligation of Esophageal Varices by the Transabdominal Route, New Eng. J. Med. 255:677, 1956.

65. Welch, C.S., Kiley, J.E., Reeve, T.S., Goodrich, E.O., and Welch, H.F.: Treatment of Bleeding from Portal Hypertension in Patients with Cirrhosis of the Liver, New Eng. J. Med. 254:493, 1956.

66. Westphal, K.: Uber eine Kompressionbehandlung der Blutunzen aus Oesophagusvarizen. Deutsche med. Wchnschr. 56:1135, 1930.